# Journal
## OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION



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**Vol. 131 October 1, 1957 No. 7**

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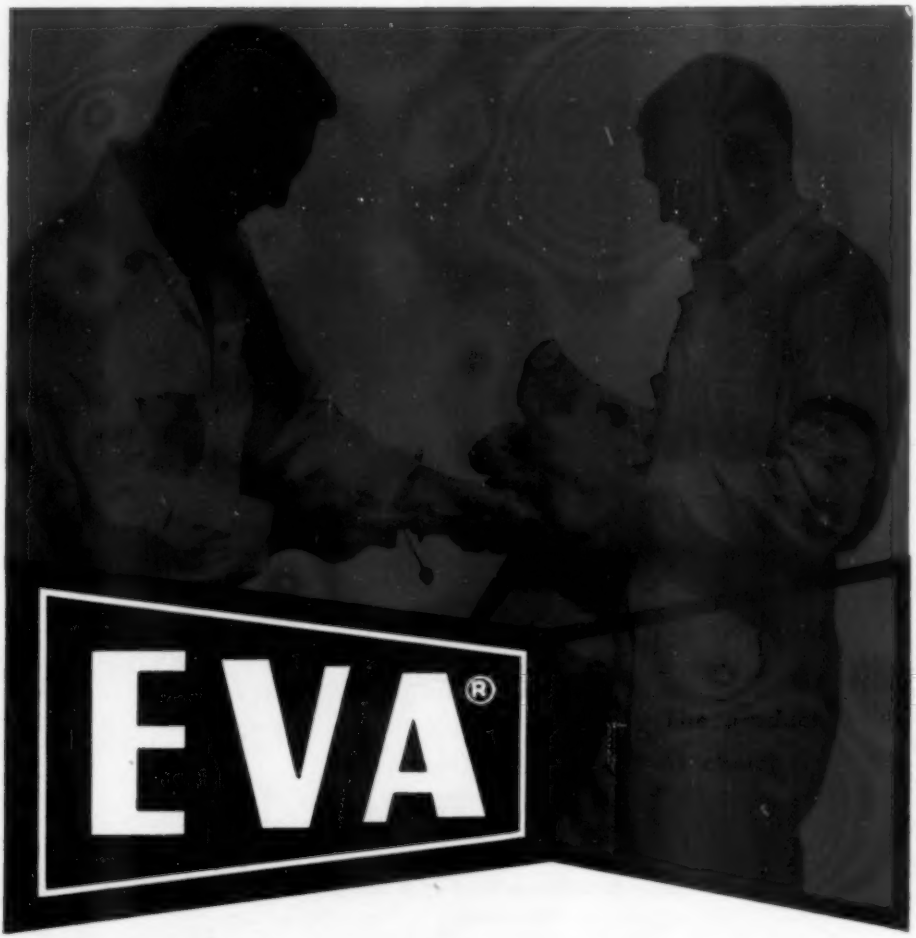


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## Correspondence

June 19, 1957

Dear Dr. Aitken:

I should like to call your attention to the fact that the last two paragraphs of the summary on the "WHO Expert Committee Rabies Report (page 492, June 1 issue, *J.A.V.M.A.)* was loosely abstracted. I do not understand why the word "even" was used in stating that puppies and kittens under 3 and 4 months of age respectively will occasionally succumb to LEP Flury vaccine.

With reference to the use of Flury vaccine in adult cats, the statement implying superiority of nervous tissue vaccines over Flury vaccines should be modified to indicate that this protective superiority was observed in an experiment which covered a period of only *70 days between vaccination and challenge* (page 13, WHO report). Regarding the *practical* use of rabies vaccine in cats, the WHO report states (p. 15-16) that cats over 6 months of age may be effectively immunized with either nervous tissue or chicken embryo vaccine although the duration of immunity in this species is unknown.

Also, regarding the handling of animals bitten by rabid animals, as an alternative to immediate destruction or six months' isolation of unprotected animals, the abstract does not mention that these unprotected animals which received post-exposure treatment should be confined in a kennel for three months (page 17, WHO report).

Sincerely yours,
s/Ernest S. Tierkel

[(1) The word "even" should not have been used; (2) the recommendation for confinement of exposed and treated animals for three months after vaccination should have been mentioned.—ED.]

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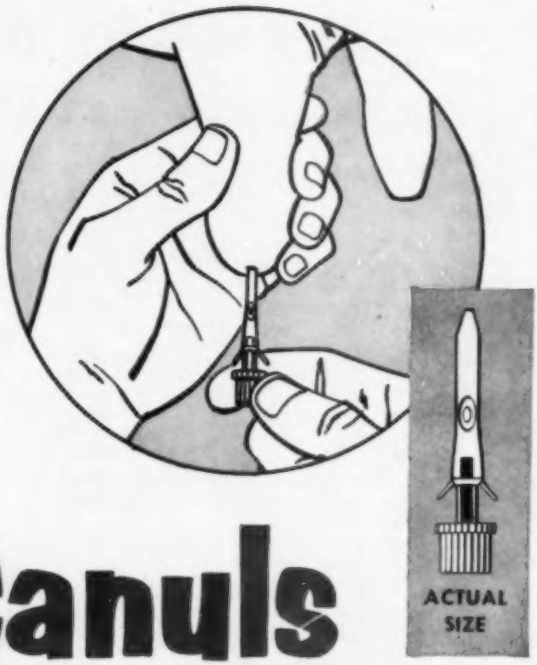


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# AVMA ☆ Report

## Gathering of AVMA Past-Presidents at Cleveland Convention

Nine of the 16 living past-presidents of the Association gathered at a luncheon meeting in the Hotel Cleveland on August 21. In the order of their terms of service they were:

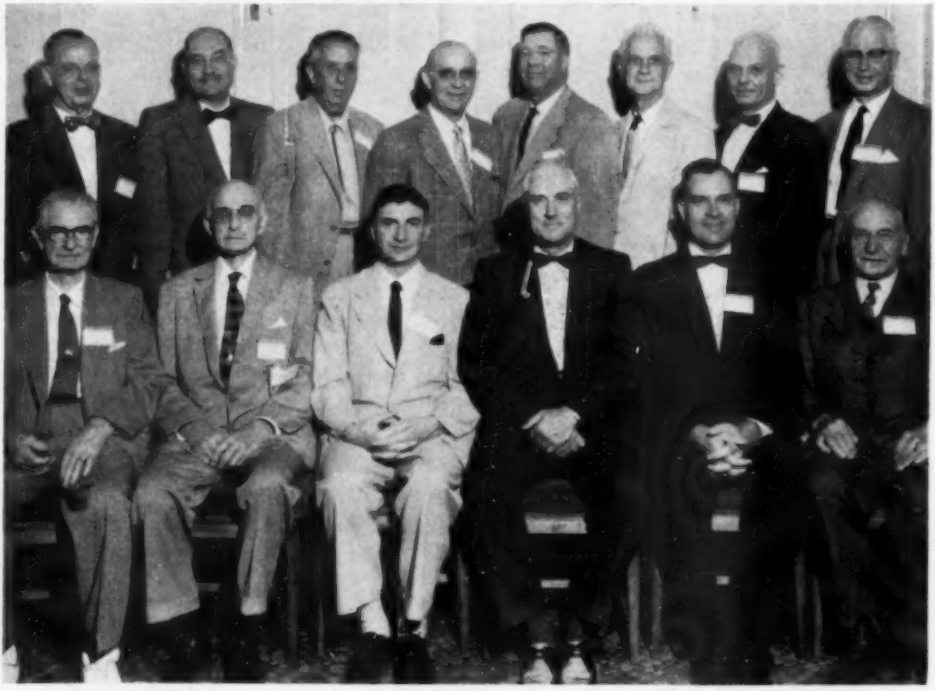
T. H. Ferguson (Wisconsin)—1929-1930
R. S. MacKellar (New York)—1934-1935
C. W. Bower (Kansas)—1943-1944
W. A. Hagan (New York)—1947-1948
C. P. Zepp, Sr. (New York)—1949-1950
W. M. Coffee (Kentucky)—1950-1951
J. R. Wells (Florida)—1951-1952
J. A. McCallam (District of Columbia)—1953-1954
A. H. Quin (Missouri)—1954-1955

Others present included President Kester, President-Elect Armistead, Executive Board Chairman Jones, former (1932-47) Board member A. E. Cameron, and Executive Secretary Hardenbergh.

President Kester presided at the informal gathering which had been arranged at the suggestion of Drs. Bower and Quin. He commented on recent AVMA developments, such as the proposed new Constitution and Bylaws which will be acted on in 1958, some of the recommendations in his Opening Session address, and the "blueprint" for the expansion of Association activities and services.

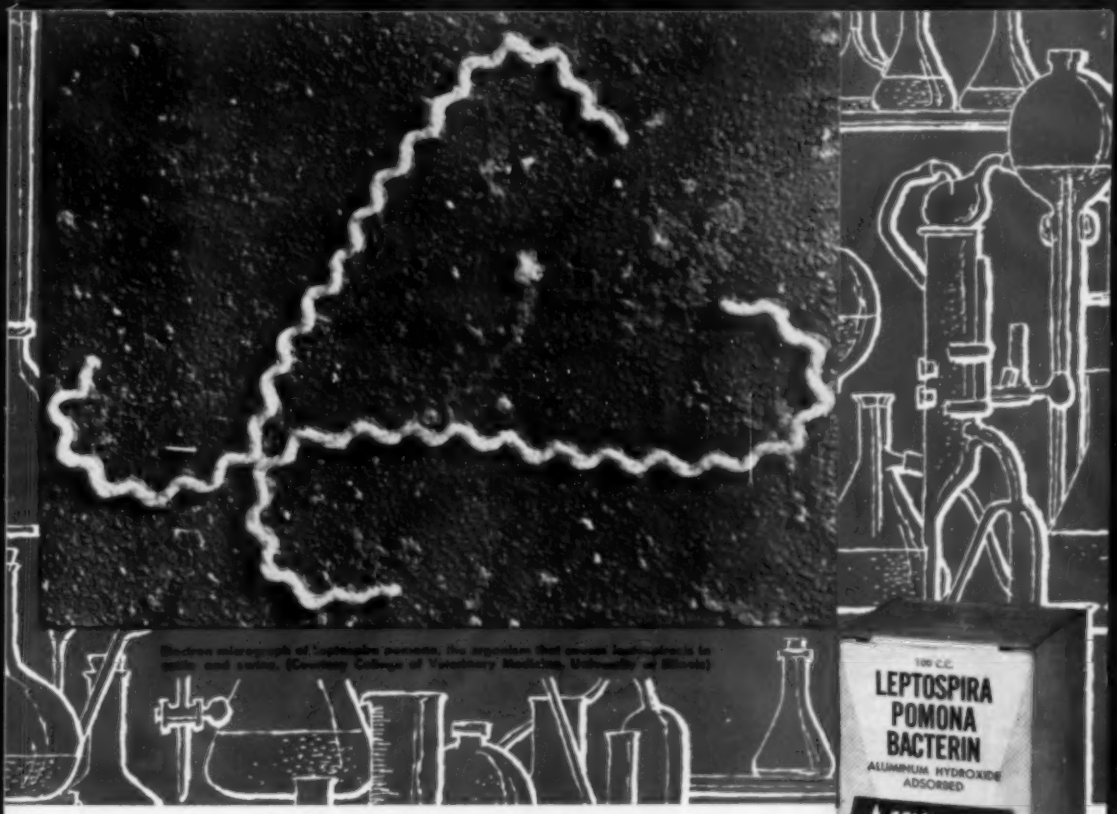
As a result of discussions of these developments, it was the consensus that AVMA past-presidents might perform a useful service to the Association by acting as an informal advisory group to the Executive Board. It was agreed that the immediate past-president should serve as its chairman, that the gathering of past-presidents should be an annual affair, and that a report from the group should be an item on the agenda of the annual meeting of the Executive Board.

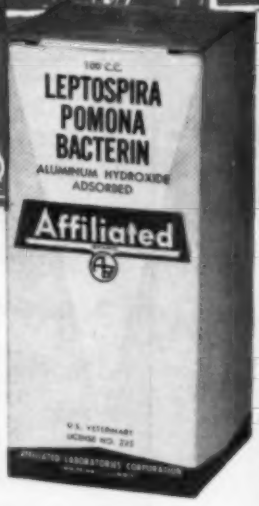
### Past-Presidents of the AVMA



Seated—L. to R.: R. S. MacKellar, Sr. (1934-1935), T. H. Ferguson (1929-1930), T. Lloyd Jones (Chairman, AVMA Executive Board, 1957), W. O. Kester (1956-1957), W. W. Armistead (1957-1958), A. E. Cameron (Member, Executive Board, 1932-1947).

Standing—L. to R.: W. A. Hagan (1947-1948), C. W. Bower (1943-1944), A. H. Quin (1954-1955), C. P. Zepp, Sr. (1949-1950), W. M. Coffee (1950-1951), J. R. Wells (1951-1952), J. A. McCallam (1953-1954), J. G. Hardenbergh, Executive Secretary.



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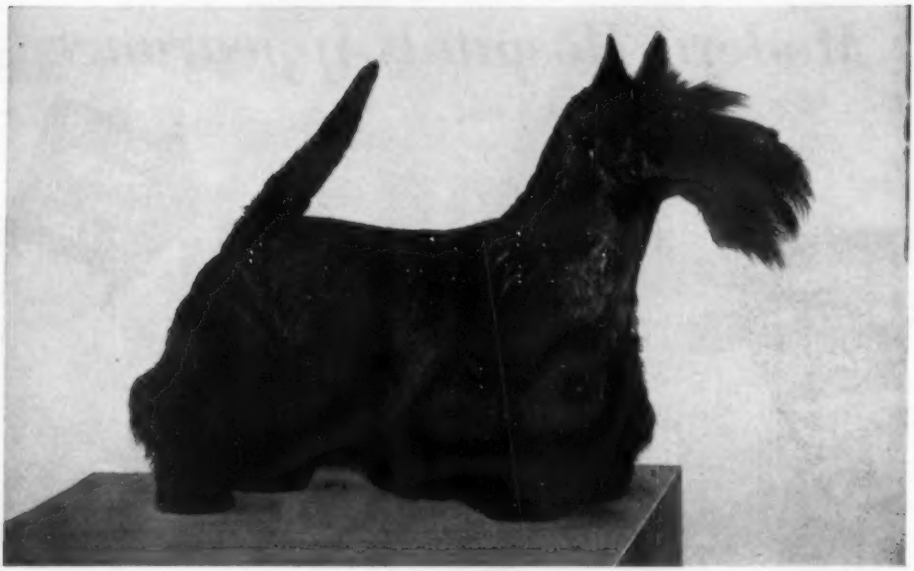

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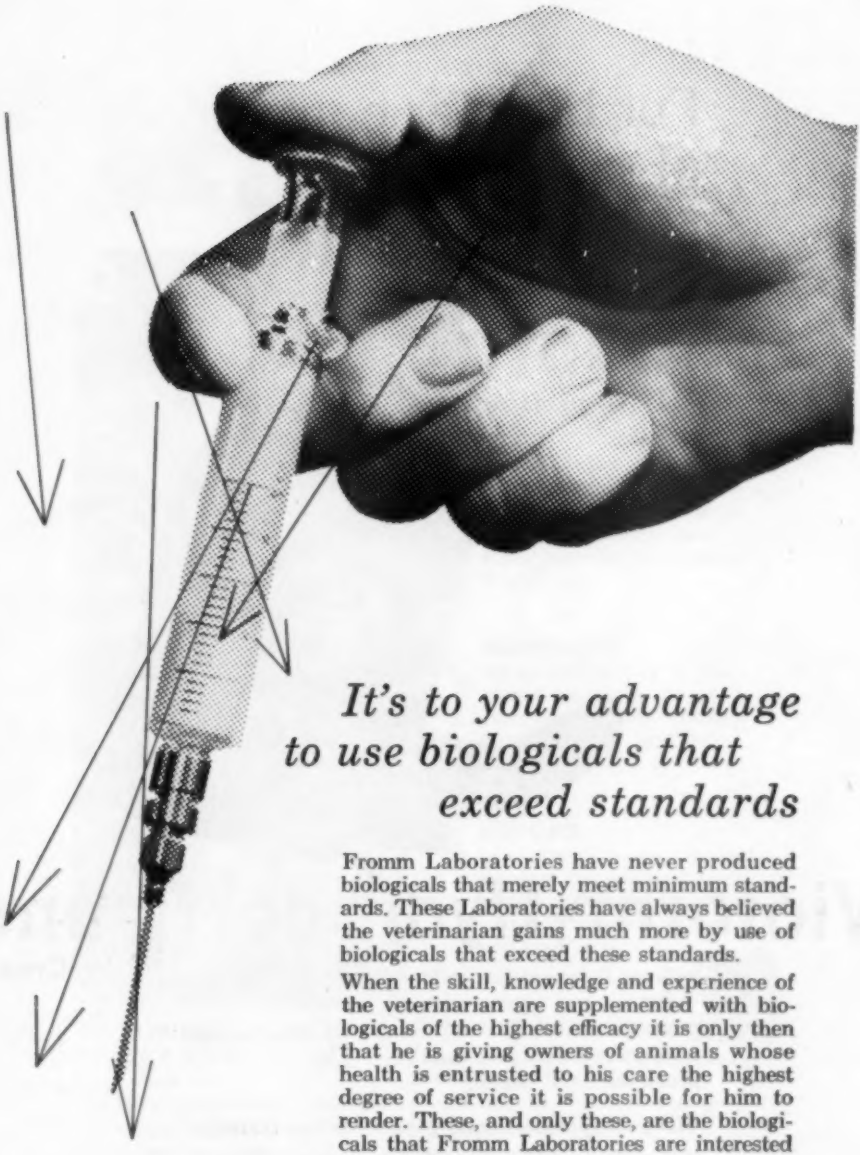

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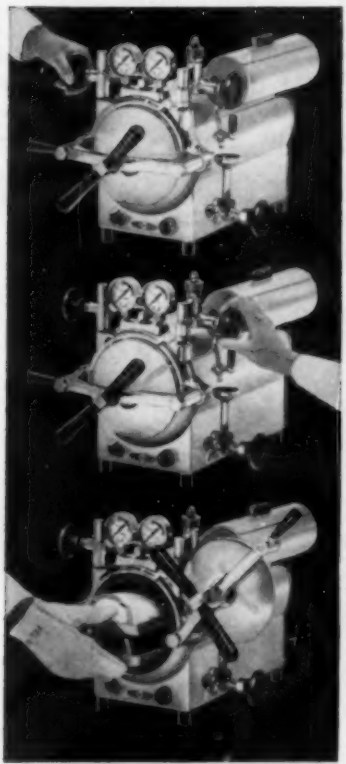

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*Jones, S. V.; Belloff, G. B., and Roberts, H. D. B.: Vet. Med. 51:413 (Sept.) 1956.

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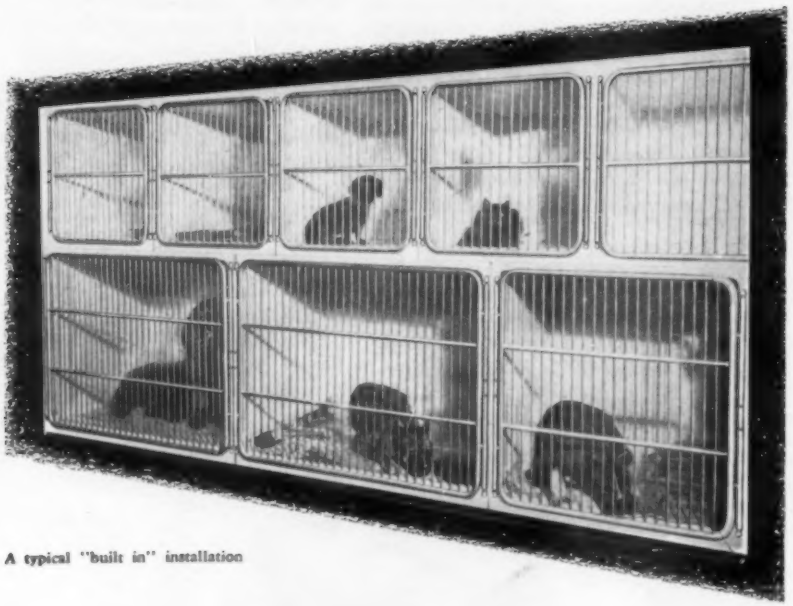

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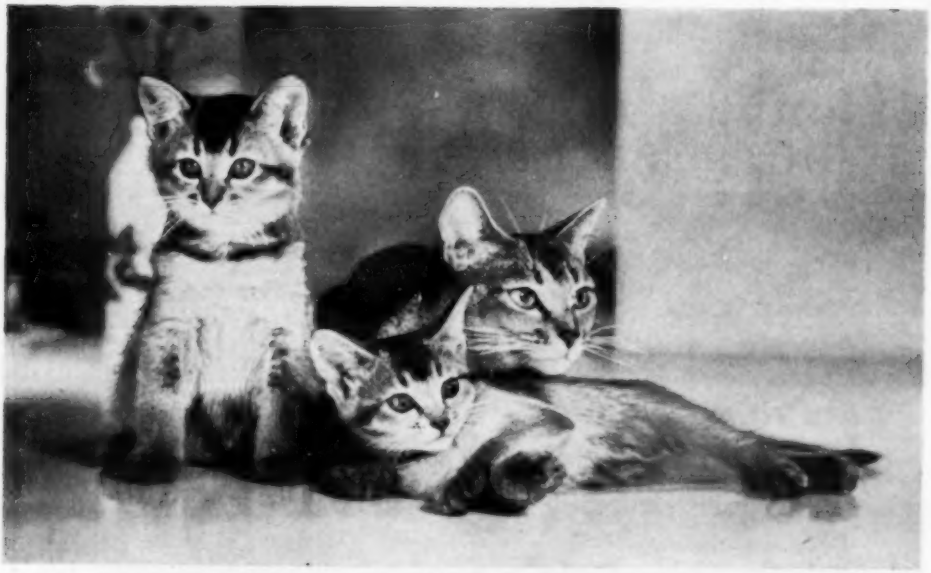

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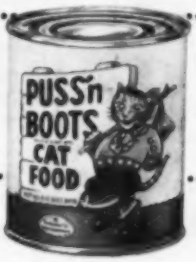


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# VETERINARY X-RAY NEWS

No. 3

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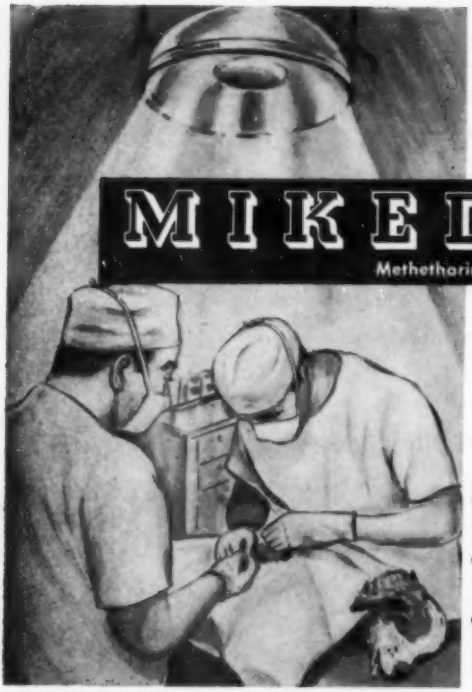

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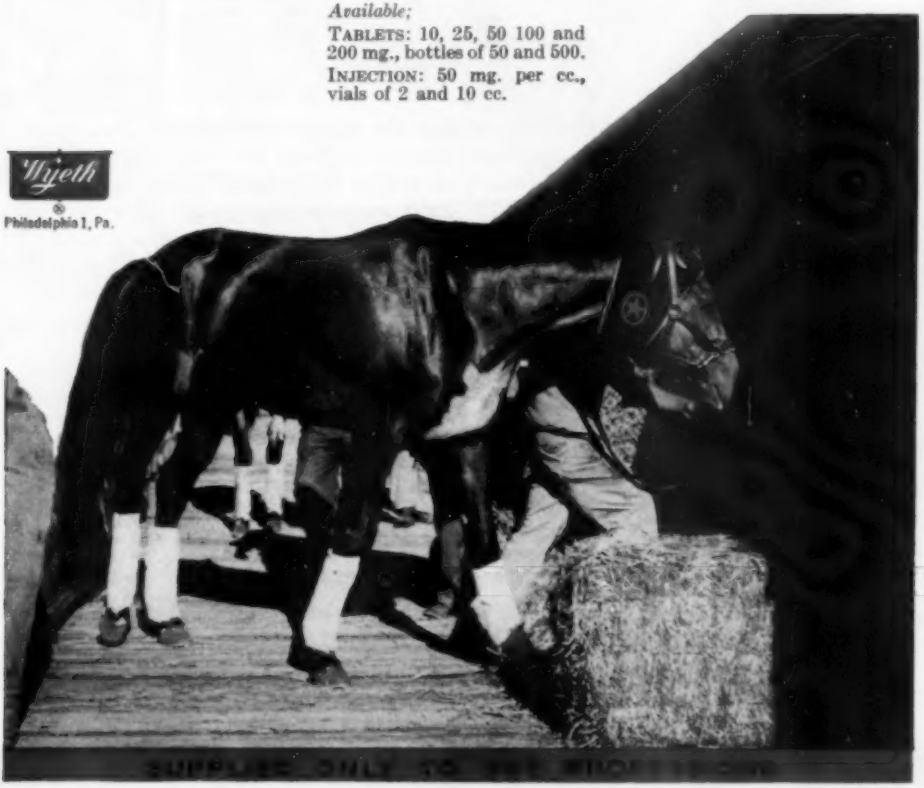
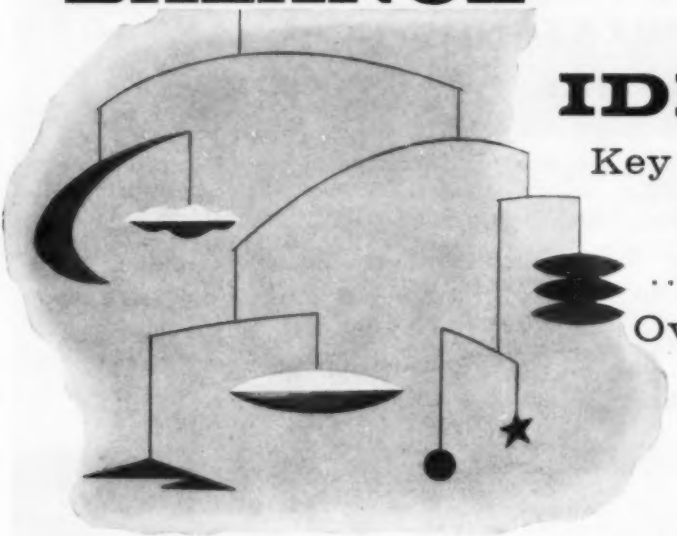


# TOTAL BALANCE



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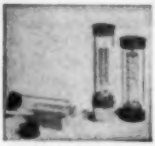

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# Canine Glaucoma. I. Methods of Diagnosis*

W. G. MAGRANE, D.V.M., M.Sc. (Med.)
*Mishawaka, Indiana*

GLAUCOMA, whether it be in man or animals, is not, in itself, a disease entity. Instead, it consists of a variety group of conditions which have as their common feature an abnormal elevation of intraocular pressure. This group may be referred to as the glaucomas.

Normal intraocular pressure is that which is compatible with the continued health and function of the eye. In the dog, however, we must give more emphasis to statistical averages in our computation of normal pressure because of our inability to perform perimetry and other exact visual function tests.

A distinction has been made between the use of the terms "pressure" and "tension" in relation to the eye.[6] The term "intraocular pressure" refers to the pressure of the intraocular fluid within the eye which can only be measured by means of a manometer. The "tension" of the eye is a measure of the state of tension of its tunics, being estimated either digitally or by means of a tonometer. In addition to intraocular pressure, tension depends upon the thickness and rigidity of the tunics, the surface area, and probably other factors.

Manometry is not practical for use in clinical veterinary ophthalmology, whereas digital palpation and tonometry are. In this discussion of canine glaucoma, then, the term "tension" will be used to denote rise and fall of intraocular pressure.

## TONOMETRY

Tonometry is defined as the measurement of the impressibility of the tunics of the eye by deforming forces applied to these tunics.[6] The impressibility depends on the resistance of the eyeball which, in turn, depends on the intraocular pressure. Thus, tonometry gives a measure, however approximate, of the intraocular pressure.

In digital tonometry, the two index fingers are placed upon the dog's upper lid. By alternating pressure on the globe from one finger tip to the other, pressing more downward than backward, one can get some idea of the degree of tension by comparison with the other eye, if normal, or with a healthy eye of another dog. By way of comparison, a glaucomatous eye will feel like a hard apple, while an eye with normal tension will feel approximately like a soft gelatin worm capsule. Digital tonometry is especially useful when the intraocular tension is either quite low or quite high, and should be the means of measurement employed when the corneal epithelium is abnormal or eroded, or when a refractory dog is examined.

Instrumental tonometry, by far the more accurate means of measuring intraocular tension, should be used when possible. The tonometer,* by movement of a needle upon a scale, records resistance offered to definite weights used to produce an indentation of the cornea. Readings as low as 16 and as high as 30 mm. of mercury may be considered within the normal range in the dog.[4]

In cooperative animals, readings can be made following two instillations, separated by a five-minute interval, of topical anesthetics. Opthaine† (0.5%) was used

Dr. Magrane is a small animal practitioner in Mishawaka, Ind.

*Abstracts from the thesis accepted by the faculty of the Graduate School of Medicine of the University of Pennsylvania in partial fulfillment of the requirements for the degree of Master of Medical Science in ophthalmology.

*The tonometer used in this work was the model of Schiötz.

†2-diethylaminoethyl 3-amino-4-propoxybenzoate hydrochloride (Squibb).

since it produces no undesirable side effects.[3]

The dog should be placed on a table in a sitting position. An assistant, or the owner, restrains the animal's head in such manner that the eyes are directed upward, the lids are separated, and the tonometer is allowed to rest by its own weight upon the center of the upturned cornea. In the separation of the lids, undue pressure must not be exerted on the eyeball itself, thus influencing the reading. The nictitating membrane often proves a deterent to the accurate placement of the footplate of the tonometer and many readings will, of necessity, be taken with the footplate between the center and upper limbal area. The degree of accuracy achieved in man with the use of the tonometer can not always be attained in the dog but, with patience, a good approximation of tension is almost always attainable. An instillation of a suitable ophthalmic antiseptic or antibiotic preparation to the tested eye is indicated, following the procedure, to forestall a possible infection from the footplate or from its abrasive action on the cornea. The tonometer itself must be cared for as a delicate instrument, including cleaning following each use if it is expected to give consistent and accurate readings.

## CLINICAL SIGNS

The diagnosis of glaucoma in the dog is relatively simple since the animal is usually presented for an examination only when the clinical signs are exaggerated and visible to the owner. Important early signs of pain and of disturbance of vision go unnoticed and, undoubtedly, in many instances the dog has been subjected to transient attacks of pressure increases before a final and permanent increase manifests itself. Whereas this simplifies diagnosis, it makes adequate treatment more difficult—especially as regards the preservation of vision. Too many animals have reached the absolute (blind) stage before being examined.

## CHARACTERISTICS OF GLAUCOMA

There are many general characteristics of glaucoma, all of which, of course, are not exhibited in any one animal. A great deal depends upon the nature of the attack, and whether it falls into a primary or secondary classification. The general characteristics include: (1) increase in intraocular tension; (2) pain; (3) cloudy, "steamy" cornea; (4) insensitive cornea; (5) shallow anterior chamber; (6) dilatation of pupil, with no response to light; (7) episcleral vascularization (congestion of episcleral vessels); (8) loss of vision (partial or complete); (9) buphthalmos (hydrophthalmos); (10) cupping of the optic disc; (11) atrophy of the optic disc and retina; and (12) atrophy of the iris.

Increase in intraocular tension has been discussed under tonometry.

*Pain.*—Pain is produced by pressure and pull on the ciliary nerves in the eye. In obtaining case histories, some owners will volunteer the information that the dog has exhibited pain, which is often displayed by the animal rubbing the eye on the rug, against the owner's leg, or on furniture. Upon digital palpation, many dogs will wince; others appear to suffer little or no pain. Pain seems to depend somewhat upon the duration of the attack—the longer the period of increased tension, the less pain—but some dogs evidence little or no pain at any time. However, after having an increased tension for a year or more, some dogs change completely in disposition and become extremely refractory. When the tension is relieved, they usually again become docile, a gratifying aftermath.

*Cloudy, "Steamy" Cornea.*—This is manifested most prominently in acute congestive glaucoma, less so in the chronic and absolute stages. The cloudiness has been attributed to an edema of the substantia propria and a collection of fluid under and between the epithelial cells.[7]

When tension is decreased by medical or surgical means, the cornea quickly clears unless the eye has been under extreme pressure for a long period. In some dogs, the fundic detail is revealed on ophthalmoscopic examination; in others, because of the cloudy media, this is not possible. In several instances, increased tension over a long period has resulted in a striate keratitis. Rucks in Descemet's membrane, due to edema and swelling of the substantia propria, causes finger-like processes or streaks in the cornea. These opacities tend to remain permanently, even after the tension has been rendered normal by surgery.

*Insensitive Cornea.*—Increased tension may cause the cornea to become insensi-

tive from pressure upon nerve filaments. In the dog, when a glaucomatous eye is brushed with a feather or wisp of cotton, there will be no reflex blinking as in the normal eye.

*Shallow Anterior Chamber.*—In acute congestive glaucoma, the anterior chamber is often shallow, due to the anterior displacement of the iris and lens. The cause of this is probably an increase in volume or swelling of the vitreous body, accompanied by an edema and swelling of the ciliary processes.[5] The chamber depth in the dog can be approximated, and compared with the normal eye, by observing the eye from the lateral aspect with penlight and binocular loupe. When the observer's eye becomes "practiced" to depth perception, shallow chambers can readily be seen when viewing the eye from the front. Whereas a shallow anterior chamber is usually associated with primary acute attacks of glaucoma, it may be observed in instances of secondary glaucoma, e.g., types of lens subluxation, swelling of the lens in cataract formation, and in iris *bombé*.

*Dilatation of the Pupil.*—Probably the most constant and significant clinical sign of glaucoma in the dog is full dilatation of the pupil with no response to light stimuli. This is also a constant finding in progressive retinal atrophy and allied conditions[2] and, therefore, should never be used as an independent entity to establish a diagnosis of glaucoma.

The dilatation may be caused by a pressure paresis of the ciliary nerves, a paresis of the sphincter fibers because of edematous pressure, or an irritation of the sympathetic nerves. If tension is relieved early enough by medical or surgical intervention, the pupil may again become normally mobile and contracted. If the attack is prolonged and has produced synechias (adhesions) at the root of the iris, or if atrophy of the iris has occurred, the pupil may remain dilated for life, even though the tension is normalized. The pupil dilatation is often one of the first signs noticed by the owner, as it produces a peculiar greenish blue hue (especially in Cocker Spaniels with acute congestive glaucoma). This color is thought to be an optical effect due to the turbidity of the optical media whereby the lens reflects light.[5]

*Episcleral Vascularization.*—Episcleral vascularization, or congestion of the episcleral vessels, is a constant finding in tension increase. The vortex veins within the sclera are affected by the initial increase in tension. This makes it necessary for the anterior ciliary veins to take over their work, thus becoming distended in the process.

The conjunctival vessels also become injected and a mild degree of chemosis may accompany the change in the vessels. This engorgement is accentuated in the chronic and absolute stages by the inevitable globe enlargement (buphthalmos). When tension is restored to normal, the vessels tend to return to near normal size; the smaller ones disappear but seldom do any completely revert to their original caliber. The conjunctiva becomes "thinner" and even atrophic after a sustained tension increase.

*Loss of Vision.*—Loss of vision, while due partly to failure of the owner to recognize the early stages, may also be due to failure on the part of the veterinary clinician to diagnose and treat the condition properly. If seen early, when there is little or only partial loss of vision, sight can usually be preserved. When only one eye is glaucomatous, the dog may or may not show signs of blindness on the affected side. To test the ambulatory vision, a heavy black patch is placed over the normal eye (held in place with laces which encircle the opposite ear and the neck). The dog is then observed as it moves through unfamiliar surroundings. When the tension increase is of long duration, and examination with the ophthalmoscope discloses extensive cupping and retinal atrophy, this test is not performed.

In the early acute congestive phase, the blindness that ensues is often of a transient nature, for when tension is normalized, vision is restored. The initial opacification of the ocular media contributes to the reduction in the vision, but ischemic changes in the retinal blood vessels, as a result of congestive changes in the retinal tissue, may also be a contributing factor. There may also be a pressure interference with transmission of impulses along the optic nerve fibers or a pressure interference with the function of the rods and cones.[1]

In one instance, following normalization of tension, two weeks elapsed before vision returned, although the ocular media was clear for most of that period. The return of vision was certainly dramatic, as all hope for sight had been abandoned. In

other dogs, return of vision ensued within hours following tension relief, especially when this was accomplished by medical means. As a rule, the tension must be relieved within 24 to 48 hours following onset of acute congestive attacks, when tensions are 60 mm. of mercury or more, in order to prevent permanent damage and loss of vision.

*Buphthalmos (Hydrophthalmos).*—The term hydrophthalmos or buphthalmos is applied to a glaucomatous eye when, under the influence of high tension, it has increased in size. These terms are used in human ophthalmology to denote infantile (congenital) glaucoma in which, because of the softness of the tissues, the gross distention of the eyeball is the main characteristic. While this is not found in the human adult, it is a constant clinical finding in canine glaucoma, whether it be primary or secondary, probably because the canine eye contains more elastic fibers. These terms are used synonymously; the French and English preferring "hydrophthalmos," with "buphthalmos" being more commonly used in this country.

Enlargement of the globe is a gradual although often rapid process. The full dilatation of the pupil, in the early stage of tension increase, gives to the eye an appearance of enlargement beyond that which is actually present. However, within a few weeks the globe may increase in size, with the cornea increasing in all dimensions and the eyeball becoming exophthalmic. As the buphthalmos increases, the eye develops an unpleasant appearance and is subjected to other inflammatory processes.

*Cupping of the Optic Disc.*—Since the lamina cribosa, that portion of the sclera which is perforated by the optic nerve, is the most yielding area of the fibrous coat, it is pressed backward, producing a depression or cupping of the optic nerve head which is recognizable with the ophthalmoscope when the ocular media is clear. Ordinarily, this is not an early finding but it has been seen distinctly in dogs a few days following an acute attack. It is most readily seen in the blind, but clear, glaucomatous eye. For the normally myopic eye, a minus 3 lens of the ophthalmoscope is used first, with changes in diopters being made to most clearly view the fundus. In the case of subluxation or luxation of the lens, when attempting to visualize the fundus around the border of the lens, a plus 7 or 8 lens is used. In cupping, the blood vessels appear to drop over the edge of the optic disc and are only faintly seen at the bottom of the depression.

*Atrophy of the Optic Disc and Retina.*—More often than not, atrophy of the optic disc accompanies the cupping. The disc becomes grayish white and few, if any, vessels can be seen emerging from it. The retina also becomes atrophic and nearly devoid of vessels.

*Iris Atrophy.*—In the chronic and absolute types, glaucomatous changes are evident in the iris. It appears atrophic and thin, especially at the pupillary border (sphincter muscle area). In time, this area appears darker, patchy, and riddled with holes. Pigmentary deposits may be found on the anterior lens surface or free in the anterior chamber itself. In well-established cases with obscure histories, it is difficult to determine if extensive iris atrophy is the result, or the possible cause, of the tension increase.

## SUMMARY

Glaucoma, an abnormal elevation of intraocular pressure, is diagnosed by tonometry, the measurement of impressibility of the tunics of the eye by forces applied to these tunics. Both digital and instrumental tonometry may be employed in the dog.

The diagnosis of glaucoma in the dog is usually relatively simple because of the exaggerated clinical signs present. These signs include: (1) increase in intraocular tension, (2) pain, (3) cloudy, "steamy" cornea, (4) insensitive cornea, (5) shallow anterior chamber, (6) dilatation of pupil, no light response, (7) episcleral vascularization, (8) loss of vision, (9) buphthalmos (hydrophthalmos), (10) cupping of the optic disc, (11) atrophy of the optic disc and retina, and (12) iris atrophy.

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# Parasitism in Sheep on Irrigated Pastures

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DUE TO THE prolonged (1952-1956) drought, in Missouri and surrounding states, considerable interest has been indicated in the possibilities of irrigated pastures for sheep. Because there was practically no information available concerning the response of sheep to pasture irrigation under midwestern conditions, an experiment was initiated to study the problem at the Missouri Agricultural Experiment Station. The purpose of this experiment was to compare the values of different pasture crops for sheep under conditions of natural rainfall, irrigation, and irrigation plus additional nitrogen fertilizer. This is a report of some of the difficulties encountered in attempting to control stomach worms (Haemonchus) and intestinal worms (Nematodirus) during the course of the experiment.

Since the conditions herein reported were experimental, it was impossible to apply some of the recognized hygienic principles necessary for the control of gastrointestinal parasitism in sheep. However, because of the inefficiency of the control program which was used, and because the circumstances of this experiment could be duplicated under natural conditions during extremely wet years, it appears that the results may be significant.

Little information is available concerning the bionomics of internal parasites of sheep under irrigated pasture conditions. In a study with *Trichostrongylus axei* infections in sheep in southern California, it was concluded that the destructive force of such natural adverse factors as desiccation, high temperature, and sunlight would be greatly reduced under irrigated pasture conditions.[5] In Nevada, workers found that on irrigated pastures the phenothiazine-salt mixture, combined with periodic therapeutic treatments, usually kept stomach worms under control.[4] Observations made in southern New Mexico indicated that Haemonchus larvae die off rapidly on irrigated pastures.[1] In the same state, investigators found that pasture rotation and preventive treatment with phenothiazine and salt kept lambs normal and healthy on irrigated pastures. The number of parasite eggs discharged by these lambs was only about 5 per cent of the number discharged by untreated lambs.[6]

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Contribution from the Missouri Agricultural Experiment Station, journal series No. 1751; approved by the director.

## MATERIALS AND METHODS

In this experiment, nine ½-acre plots were seeded for pasture (table 1). Plots 1, 6, and 7 were not irrigated. Plots 3, 4, and 9 were selected for routine studies of the parasite problem as well as it could be determined by the worm egg discharge in the feces.

TABLE 1—Seeding Order of Experimental Pasture Plots

| Orchard grass; Ladino clover | Orchard grass; Korean lespedeza | Orchard grass only |
|---|---|---|
| Plots 1, 2, 3 | Plots 4, 5, 6 | Plots 7, 8, 9 |

The season's water was supplied by overhead sprinklers, in ten separate applications, at the rate of 2 inches per application. The ewes and lambs were placed on pasture April 25, and on July 7 the lambs were weaned and the ewes were removed. On October 1, the lambs were moved from the experimental plots to drylots.

All ewes were drenched with phenothiazine the day before they were placed on experimental pastures. Phenothiazine-salt mixture (1:10) was available to the ewes and lambs at all times. The particle size of the drug was well within the range considered most efficient. When it became evident, on June 1, that radical measures would be necessary for parasite control, standard sized therapeutic doses of phenothiazine, or copper sulfate-nicotine sulfate (Cu-Nic), were administered at two-week intervals.

As far as could be determined, the phenothiazine-salt was consumed in average amounts, although the lambs did not appear to eat much until they were near weaning age.

To study the immature parasitic larvae present, grass clippings were cut at random over similar areas of three different irrigated pastures. A kilogram (2.2 lb.) of grass from each pasture was placed in a large Baermann funnel and covered with warm water. At the end of two hours, 300 cc. of water was withdrawn from the funnel, the nematode larvae from 3 aliquots were counted, and the average was used for calculating the total num-

TABLE 2—Nematode Larvae Counts Made from Irrigated Sheep Pastures

| Date | Larvae recovered from kilogram of grass | | |
|---|---|---|---|
| | Plot 3 | Plot 4 | Plot 5 |
| July 6 | 51,000 | 16,000 | 11,750 |
| Aug. 24 | 3,000 | 2,250 | 1,450 |
| Sept. 19 | 5,000 | 1,850 | 2,100 |

ber. A high percentage of the larvae concentrated in this manner were larval stages of *Haemonchus contortus*. The methods used for larval concentration during the study were rather crude, with a probability of considerable error,[2] but the counts (table 2) give a good estimate of the immature parasite populations on these irrigated pastures.

## RESULTS

During the early months of the study, the worm egg output in the lambs was relatively light. Counts made by the Stoll dilution method varied from 400 to 1,000 eggs per Gm. (EPG) in most lambs. However, during July and August the counts increased to from 3,000 to 10,000. Usually a marked rise in egg count could be observed at the end of the second week following treatment.

At the beginning of the study, the EPG counts in the ewes were relatively low, usually 200 to 600, but late in June they rose sharply. In some ewes, Haemonchus counts were 20,000 to 30,000 EPG and Nematodirus counts were 1,000 EPG. In a few instances, large numbers of Strongyloides were also found.

The signs of heavy parasite infection, such as marked anemia, edema, weakness, anorexia, and frequent scouring, were more severe in lambs on irrigated plots. Death losses in the lambs were relatively light until September (table 3), the greatest losses occurring in September and early October. By then it was evident that the worm burden was breaking the resistance of practically all animals on the irrigated plots.

TABLE 3—Losses Due to Parasitism While on Pasture

| | Irrigated plots | | Nonirrigated plots | |
|---|---|---|---|---|
| | No. lost | Per cent | No. lost | Per cent |
| Lambs | 15 | 20.0 | 0 | 0 |
| Ewes | 5 | 8.0 | 2 | 2.7 |

Losses of ewes due to parasitism did not occur until late in June. However, before removal from the experimental plots (July 7), 7 ewes (11%) died as a result of severe Haemonchus infections and only 2 of the 7 were from nonirrigated pastures.

## NECROPSY FINDINGS

The pathological changes appeared to be essentially those characteristic of pure *H. contortus* infection, although in most lambs considerable numbers of Nematodirus spp. were found. Both the ewes and lambs were extremely anemic. The hemoglobin level just prior to death was usually about 30 to 40 per cent of normal. Edema (bottle jaw) was observed in all lambs and an excessive accumulation of straw-colored fluid was present in the peritoneal cavities of all ewes examined. The worm burden of lamb 76 (plot 9), which appeared about average for those necropsied, was: *H. contortus*, 9,500 (approx. 70% immature); Nematodirus spp., 1,500 (the majority mature).

## DISCUSSION

Because of the rapid growth of orchard grass and legumes when they were irrigated, it was impossible to rotate the sheep from a pasture for as long as two weeks. The vegetation soon became tall, coarse, and unpalatable so that the sheep would graze only in small, isolated patches.

Even though pasture rotation was not possible, it seemed unusual that such heavy parasite infestations developed in lambs on pasture in spite of the phenothiazine control program. These findings are not totally in agreement with those of workers in New Mexico[1] and Nevada[4]. Probably the different results were due to the high relative humidity and somewhat cooler spring and summer temperatures of the central Missouri area. It is of interest that the incidence of pasture infestation was much higher in early July than August and September when it was unusually dry and hot.

Other possible explanations for this experience are: Much of the heavy pasture infestation with stomach worm larvae could have occurred in May and June before the lambs began eating sufficient phenothiazine to curtail parasite egg production and subsequent larval development; also, the strain of Haemonchus involved may be partially resistant to the effect of phenothiazine.[3] Ecological factors favorable to parasites, such as those produced by irrigation, would be an aid to the development of drug tolerance.

## SUMMARY

In an effort to control gastrointestinal parasitism in sheep, under irrigated pas-

ture conditions, a phenothiazine-salt mixture was fed continuously, and therapeutic doses of phenothiazine or Cu-Nic were given biweekly but failed to give adequate control. Twenty per cent of the lambs and 11 per cent of the ewes were lost due to parasites. Signs of severe gastrointestinal parasitism were observed in most of the animals at some time during the study. Some possible reasons for the difficulty of control are presented.

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*Iron-Dextran Therapy in Children.*—An iron-dextran complex (Imferon) was given in 1- to 2-ml. doses (50 to 100 mg. of elemental iron) daily into the gluteus muscles of 24 children with iron-deficiency anemia. Hemoglobin values rose about 11 Gm./100 ml. in three weeks, the reticulocyte response being most marked in patients with severe anemia. There were no untoward effects.—*J.Am.M.A. (June 29, 1957): 962.*

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*Tuberculin Testing of Children.*—The tuberculin test is the most reliable and inexpensive means for early discovery of primary tuberculosis. Tuberculin patch-testing of all children every three months, by the mother in cooperation with the physician, has been suggested.—*A.M.A. J. Dis. Child. (April, 1957): 396.*

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*A Rabid Bat.*—A boy in Oklahoma was bitten on the finger when he attempted to pick up a hoary bat (*Lasiurus cinereus*), which was on the ground (with 2 young attached to her) and unable to fly. When placed in a wire cage, the bat attacked and tore to pieces one of her young and, in 28 hours, she was dead. Her brain showed no Negri bodies, but inoculated mice showed typical rabies and their brains were heavily loaded with Negri bodies. The boy is receiving treatment.—*Morbidity and Mortality Release, Aug. 2, 1957.*

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A vaccine against tularemia is being tested on prisoners in an Ohio penitentiary. Prompt streptomycin therapy brought quick recoveries to those who developed infection.—*Sci. News Letter (June 22, 1957): 386.*

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*Dog Population in Europe.*—The dog population in eight European countries has been estimated to total over 2.1 million. Of the 20 breeds listed, the most popular were German Shepherds, Cocker Spaniels, Dachshunds, Pointers, Boxers, and Poodles. —*Gaines Dog Res. Center Bull., Aug., 1956.*

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*Nightshade Poisoning.*—In a dry season, when green feed was scarce, tomato vines (a member of the nightshade family), which were not bearing fruit were thrown into a cattle yard. Soon, 8 calves and 2 cows were dead. The stems, leaves, and roots of the tomato plant are definitely poisonous. In another instance, sprouts from seed potatoes (another nightshade), were fed to a sow. She and her pigs were soon dead.—*A. A. Case in Sheep Breeder (Aug., 1957): 10.*

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On June 30, 1957, garbage was being fed on 11,368 premises to 1.25 million swine, only 5.6 per cent of which were fed raw garbage. Most of the latter were in four states: Texas, 2,710; Massachusetts, 4,270; Connecticut, 14,496; and New Jersey, 43,-136. In 29 states, all garbage-feeding establishments were inspected monthly and in five other states, 98 per cent or more were similarly inspected.—*U.S.D.A. Rep., June 30, 1957.*

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Vermont has been declared modified certified brucellosis-free, the fourth state to achieve this status this year. The other states so certified are Connecticut, Delaware, Maine, Minnesota, New Hampshire, North Carolina, Washington, and Wisconsin. Other areas so designated include 440 counties in 27 other states and in Puerto Rico.—*U.S.D.A. Release, Aug. 30, 1957.*

# Clinical Data

## Pneumonia in California Lambs

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APPROXIMATELY 2,000,000 lambs are fattened annually in California. The majority of these lambs originate from the range country of California, Oregon, Washington, Nevada, and the Rocky Mountain states and are finished on irrigated clover pastures in the central valley districts of the state.

One of the most serious disease problems among these lambs is pneumonia. This paper presents clinical data, pathological and bacteriological findings, and slaughterhouse observations concerning this disease.

### CLINICAL DATA

The clinical picture is much the same as previously described.[3,5,7] There are usually a few sudden deaths, with no premonitory signs, one to three weeks after shipment to the irrigated pasture, followed by a variable number of lambs showing depression and gauntness. The nasal secretions may or may not increase.

If untreated, many of the lambs first affected die in one to three days, but as the disease progresses a more persistent course evolves, with some apparent recoveries and a variable number of so-called "chronic" cases. These latter animals rarely recover sufficiently to make economic gains. Mortality has varied from 0.5 to 40.0 per cent and the disease may be rapidly self-limiting or it may continue for several weeks.

Feeder lambs of some geographic origins are reputed to be more susceptible to pneumonia than others. One such area is the northern coastal section of California, which yearly produces 300,000 to 400,000 feeder lambs of variable quality. Because of the prevalence of pneumonia in these lambs following movement to irrigated pastures, they are routinely purchased at prices below the average for feeder lambs from other areas.

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The authors thank Miss Margaret Meyer for bacteriological work.

In 1956, the California State Department of Agriculture made a survey of 195,832 lambs on irrigated pasture to determine the mortality from pneumonia in flocks in which the disease appeared (table 1).

TABLE 1—Mortality from Pneumonia Among Clover-Fed Lambs According to Area of Origin

| Origin of lamb | No. of lambs in survey | Mortality (No.) | (%) |
|---|---|---|---|
| Northern coast, Calif. | 42,640 | 2,132 | 5 |
| Central Valley, Calif. | 4,500 | 45 | 1 |
| Northeast Calif., Nev., and Utah | 148,342 | 1,681 | 1 |

### GROSS PATHOLOGICAL FINDINGS

A quantity of excellent pathological material for study was afforded in the northern Sacramento Valley, where some 30,000 north coast feeder lambs were being fed. During the summer feeding period, approximately 1,500 lambs died, 500 of which were presented for necropsy. With few exceptions, pneumonia was considered the primary cause of death.

The pathological changes were studied in 96 unselected lambs with pneumonia, and bacteriological samples were collected from 50 of these. Blocks of lung tissue were taken for sectioning at the margins of the most advanced lesions. Because of the large number of lambs examined, only the changes in the lungs will be discussed.

Diffuse pulmonary congestion was observed in 14 lambs. The lungs were diffusely inflamed, swollen, and edematous (fig. 1A). The trachea and bronchi contained white froth. Red consolidation was observed in the apical, cardiac, and intermediate lobes and in the anteroventral part of the diaphragmatic lobes of the lungs of 29 lambs. The consolidated portions were dark red, swollen, firm, and well defined (fig. 2). Gray consolidation was found similarly distributed in the lungs of 11 lambs. The tissue was gray, friable, and sometimes emitted a foul odor. On the lungs of the remaining lambs, there were extensive fibrinous adhesions to the chest wall which, in some cases, partially enveloped the peri-

Fig. 1—Lungs of lamb showing gross diffuse congestion (A) and advanced pleuritis (arrows) involving most of the right side (B).

cardium. These fibrinous areas were usually bilateral and anteroventral but sometimes involved one entire lung (fig. 1B). Foul-smelling, gray, liquid exudate was sometimes present in the pleural cavity. Under the pleural fibrin, the lungs of 37 lambs showed diffuse congestion, while those of 5 lambs showed red consolidation.

## MICROSCOPIC FINDINGS

Microscopically, the lung changes could be divided into four stages, based on a comparison of the condition of the oldest part of the lesion with each successive zone of more recent development.

*Stage 1, Inflammatory Edema.*—This was found as a diffuse change in 14 lambs but, when parts of the lungs were consolidated, it was represented by a zone at the advancing margin of the lesion. The septal capillaries were dilated and filled with blood, and homogeneous, pink fluid was found in the alveolar lumina. The septal cells showed proliferation, were rounded, and protruded into the alveolar spaces or floated free in the fluid (fig. 3). Few neutrophils were present in the alveoli, but many were present in the septal capillaries.

*Stage 2, Leukocytic Exudation.*—This stage, found in the lungs of 44 lambs, was characterized by focal areas of alveoli filled with neutrophils (fig. 4). Alveolar edema and septal cell proliferation were still present but were masked by the leukocytic exudate. The advancing margins of these foci showed inflammatory edema (stage 1). In many cases, the neutrophils in the central parts of these foci were necrotic. In 16 of the 44 lambs, alveolar septa were also necrotic, and this resulted in massive areas of complete necrosis. On the lungs of 5 lambs, there was pleural fibrin over the consolidated portions.

*Stage 3, Fibrinous Stage.*—This stage, found in only 1 lamb, was seen in areas where alveolar leukocytes had disappeared, leaving alveoli filled with thick, coarse fibrin.

*Stage 4, Resolution.*—This stage, which

Fig. 2—Anterodorsal view of a lamb's lungs showing red consolidation in anteroventral lobes.

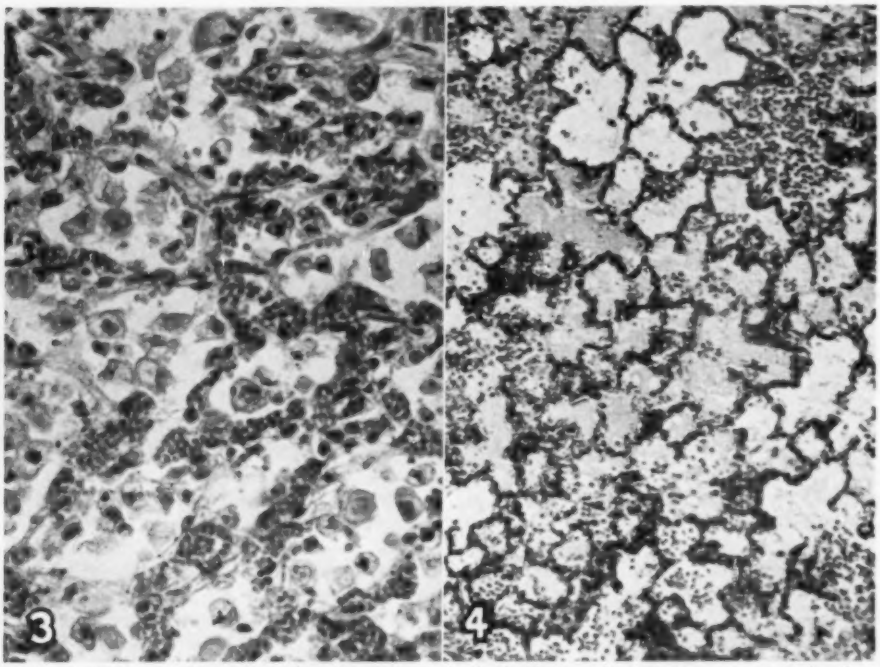


Fig. 3—Stage of inflammatory edema in the lung of a lamb. The septal capillaries are congested and numerous septal cells fill the alveoli. x 600.

Fig. 4—Stage of leukocytic exudation in a lamb's lung. The alveoli contain neutrophils and fluid. x 225.

was difficult to distinguish from the stage of inflammatory edema (the same type of exudate was present in both), was found in the lungs of 37 lambs showing chronic pleuritis. The alveoli were relatively free of leukocytes and contained mainly septal cells and fluid. The alveoli showed varying degrees of collapse and the lungs were usually hyperemic.

The pleural fibrin appeared as a thick layer separated from the underlying tissue by a zone of neutrophils. Organization of the fibrin (fig. 5) was found in all but a few of these lambs.

In addition to these microscopic changes, lungs from some of the lambs with advanced cases of pneumonia showed hemolysis of erythrocytes, edema of interlobular septa, and engorgement of the septal lymphatics with fluid. Septal fibrosis was observed in 1 lamb and abscesses in 2. Alveolar hemorrhage was rarely encountered. Bacteria were seen in the majority of lungs and, with the hematoxylin and eosin stain, appeared as purplish clumps in terminal bronchioles and alveoli. Atelectasis was seen in areas bordering consolidation and in lungs covered with organized pleural fibrin. Alveolar emphysema was never extensive. No parasites or fungi were observed.

### BACTERIOLOGICAL FINDINGS

At necropsy, the lungs and heart blood of 50 lambs were cultured. *Corynebacterium pyogenes* was recovered in pure culture from the heart blood of 13 lambs; mixed with *Pasteurella multocida* from the lungs of 10 lambs; and with micrococci, streptococci, and Pseudomonas from the lungs and heart blood of 11 lambs. Staphylococci, streptococci, and Pseudomonas in mixed cultures were isolated from the lungs of 16 lambs.

### SLAUGHTERHOUSE OBSERVATIONS

In the course of other investigations,[6] which necessitated examination of lungs from fat lambs at slaughter, the authors

became aware of a small pneumonic lesion appearing frequently in seemingly healthy lambs. Accordingly, the lungs from 12,495 fat lambs were examined at slaughter plants. All lambs had made satisfactory gains and were considered healthy by the owner. In addition, there had been no cases of pneumonia in the bands of origin during the current feeding season. The lesions (table 2) had not produced sufficient reaction to disturb the clinical well-being of the lambs.

The typical gross lesion was a firm, red, depressed area, rhomboid or linear in shape, and 1 to 4 cm. in diameter. Usually, multiple foci were present in the apical, cardiac, and intermediate lobes or the anteroventral portions of diaphragmatic lobes. Grossly, these foci were similar to areas of focal atelectasis. The bronchi leading into these areas were slightly dilated and contained small amounts of slimy, cloudy exudate. No lungworms were found.

Microscopically, the picture was one of chronic bronchiolitis and alveolar atelectasis. The bronchioles contained a mucous secretion and varying numbers of neutrophils, and the peribronchiolar solitary lymph follicles were markedly hyperplastic. The surrounding alveoli were usually atelectatic and often contained neutrophils, septal cells, and fluid. There was sharp separation between involved and normal lung tissue.

TABLE 2—Incidence of Small Pneumonic Lesions in Clinically Healthy Lambs at Slaughter

| Area of origin | Northern coast Calif. | Central Valley Calif. | Northeast Calif. | Nevada | Oregon |
|---|---|---|---|---|---|
| No. of lambs | 5,342 | 3,889 | 1,891 | 732 | 641 |
| Per cent affected (range) | 70-90 | 40-60 | 20-30 | 20-30 | 60-70 |

## DISCUSSION

An opinion prevalent among California lamb feeders that a higher mortality occurs

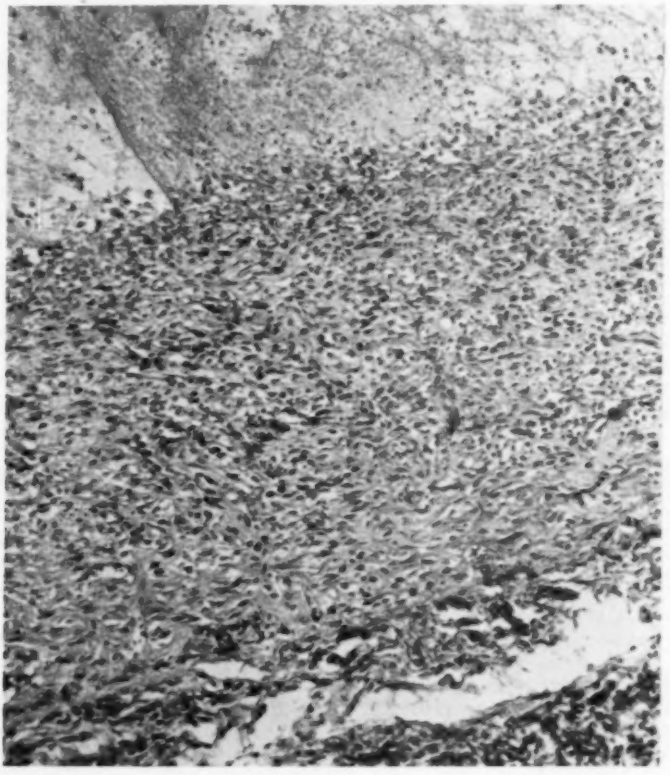

Fig. 5—Organized pleural fibrin in the lung of a lamb. The lung parenchyma is below, the fibrous layer in the middle, and loose fibrin at the top. x 225.

among feeder lambs originating from the northern coastal area of the state seems well founded (table 1). Various theories have been suggested to explain this observation. Most of these concern weather, feed, or some other environmental influence. None have been subjected to critical evaluation, but most are untenable because they depend on the primary hypothesis of a unique environmental experience. However, there is reason to believe that the environment of certain areas is more favorable than others for the perpetuation of an etiological agent and that this may be manifested in a higher prevalence of disease.

From the standpoint of the clinician, pathologist, and bacteriologist, this pneumonia has as yet to manifest any variation, other than prevalence, ascribable to the area of birth and early life of the lamb. It seems reasonable to assume, therefore, that regardless of the area of origin of the lamb, a common but as yet unknown etiological agent, or agents, exists.

Stress has long been considered a precipitating force in this type of pneumonia in lambs. Transportation, either by truck or rail, has for years been credited as a source of severe stress, and the frequent appearance of pneumonia following the movement of lambs certainly lends credence to this theory. However, this source of stress is common to the majority of lambs fed in California as very few clover-ranch operators raise their lambs. Furthermore, movement from one part of the state to another could not be considered more stressful than movement, for example, from Idaho to California.

Perhaps the most plausible explanation is that these lambs have experienced an inapparent pneumonia before being moved from their area of origin. This theory is strengthened by finding small pneumonic lesions in 60 to 65 per cent of the lungs of 234 fat lambs shipped directly to slaughter from the northern coastal section of California, compared with only 17 per cent of 365 fat lambs shipped directly to slaughter from Nevada.

It seems probable that the correlation between the percentage of lungs affected (table 2) and the percentage of mortality from pneumonia, by area of origin (table 1), is more than coincidence. It is felt that this lesion represents a primary pathological process, initiated by an undetermined etiological agent, or agents, which is not apparent during its course but is capable of becoming a focus of a more widespread secondary pneumonia following periods of environmental stress. Thus, this disease presents features similar to viral pneumonia of pigs.[1,4]

From this study, one could speculate that bacteria entered by way of the bronchi, lodged in the terminal bronchioles and alveolar ducts, and that the tissues reacted with hyperemia, edema, and septal cell proliferation. As the bacteria multiplied and spread peripherally, more lung tissue probably became involved, the older part of the lesion began to show migration of neutrophils which filled the alveoli in an ever-widening area, and inflammatory edema developed at the advancing margins of the lesion. Leukocytes in the central part of the lesion probably began to undergo necrosis which progressed peripherally, involving the alveolar septa and resulting in massive areas of necrosis. If the lamb recovered from the stages of leukocytic exudation and necrosis, the lung progressed into the fibrinous stage or into resolution but, if the infection spread to the pleura, a chronic fibrinous process probably was established.

The fibrinous stage of pneumonia was observed in only 1 lamb. Fibrin in minute amounts was probably present in all stages, but extensive alveolar fibrin, such as is seen in fibrinous pneumonia of cattle, was rarely observed. Either the lambs died in the leukocytic exudate stage, or the fibrinous stage was not a feature of this disease. Therefore, we believe that the term "fibrinous pneumonia" is unsuitable for this condition.

It was impossible to differentiate the stage of resolution from the stage of inflammatory edema. Resolution probably was present in lambs with congested lungs covered with organized fibrin, but in these cases the histological picture was almost identical with the stage of inflammatory edema.

The gross changes could be fairly well correlated with the microscopic changes, although more than one microscopic stage was present in a single section of lung, the predominant one being responsible for the gross appearance of the lung. The gross change of congestion correlated well with the stage of inflammatory edema, and in red consolidation the predominant micro-

scopic change was leukocytic invasion of alveoli with hyperemia of septal capillaries (stage 2). With large areas of lung showing ischemia and leukocytic invasion, or with massive necrosis of the lung, the stage of gray consolidation was seen grossly. Resolution, if present, was indicated, grossly, by congestion.

The lung lesions observed in "normal" lambs at the slaughterhouse were interpreted as chronic catarrhal inflammations of the terminal bronchioles, with hyperplasia of peribronchiolar lymphoid tissue. Inflammatory involvement of alveoli was secondary to the bronchiolar process and not present in all cases. Alveolar atelectasis was usually present because the bronchioles were partially occluded by mucopurulent exudate. Further studies are being made on the significance of this lesion in sheep and its relation to clinical pneumonia.

To date, the most satisfactory method of treatment has consisted of separating the visibly affected lambs by means of a dodge chute and drenching them with a sulfonamide solution (1.5 gr. /lb. of body wt.), repeated on the second, third, and fourth days if necessary. When it is possible to give the so-called "healthy" lambs one treatment of sulfonamide, this markedly reduces the spread of the disease. In any event, the healthy group should be observed daily and any affected lambs separated and placed in the hospital group.

## SUMMARY

Clinical, pathological, and bacteriological data and slaughterhouse observations are presented on a pneumonic condition in feeder lambs in California. A survey reveals that it is the most serious disease problem encountered in such lambs, and that there exists a correlation between the prevalence of this disease and the origin of the lamb.

At slaughter, the lungs of healthy lambs revealed a high incidence of a small pneumonic lesion which was highest in lambs originating from the area where the clinical manifestation of this disease was highest. Similarities seem to exist between this disease and viral pneumonia of pigs.

Of 96 pneumonic lambs examined at necropsy, the lungs of 14 showed gross congestion, 29 showed red consolidation, and 11 showed gray consolidation. Pleuritis was present with lung resolution in 37 and with lung consolidation in 5 lambs. Microscopically, the lung changes showed four stages: In 14 lambs, the predominant change was inflammatory edema, in 44 it was leukocytic exudation, in 1 the fibrinous stage was found, and in 37 resolution was present.

Bacteriological surveys revealed no significant findings other than the high incidence of *Corynebacterium pyogenes* in the affected lungs.

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*Controlling Fly Maggots on Sheep.*—It has been found, in Australia, that the best protection against fly "strike" is by power "jetting" to saturate the wool with the insecticide solution; however, jetting is arduous and time-consuming. Since some insecticides will diffuse down the wool fiber, the effects of spraying and jetting were compared on two large flocks of sheep. Surface spraying with 0.5 to 0.2 per cent Dieldrin was equivalent to jetting with 0.02 per cent of the same insecticide and was more effective than spraying with similar dilutions of Aldrin. To obtain equal results, spraying requires less total fluid than does jetting but it requires two to four times as much insecticide because of the higher concentration required. However, because of the saving in time, spraying is more economical.—*N. P. H. Graham in Austral. Vet. J. (June, 1957): 137-140.*

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Enterotoxemia, due to *Clostridium welchii* type D, was diagnosed in a calf 3 months old, in Australia.—*Austral. Vet. J. (June, 1957): 144.*

# The Control of Laboratory-Induced Cervical (Jowl) Abscesses in Swine by the Continuous Feeding of Various Levels of Chlortetracycline

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R. F. ELLIOTT, Ph.D.

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IN 1954, CERVICAL ABSCESSES, also known as jowl abscesses or feeder boils (fig. 1), ranked third as a cause of condemnation of pork, accounting for 2.6 per cent of all swine condemned on federal inspection at

Fig. 1—Photograph showing a cervical abscess in a pig.

slaughter.[5] About 1,300,000 heads and jowls of the 50,000,000 federally inspected swine were condemned because of cervical abscesses, a loss of nearly $1,000,000 to the meat packers. More recent reports from the field indicate an increase in the incidence of this disease.

The *beta* hemolytic Streptococcus is considered the principal etiological agent of cervical abscesses, although *Pasteurella multocida, Escherichia coli,* and *Corynebacterium pyogenes*[1,2,6-9] have also been isolated. An inoculum prepared from a *beta* hemolytic Streptococcus sp., belonging to Lancefield's serological group E isolated from a field case, regularly induced abscesses of the pharyngeal region indistinguishable from those seen in field cases.[3] The test organism was then reisolated from the lesions. Giving the inoculum in the feed was the most successful method of producing the abscesses, although intranasal and intrapharyngeal instillation produced similar results. Strains of *C. pyogenes* and *P. multocida,* isolated from similar lesions, did not produce abscesses or any other ill effects when fed in a similar manner.

The experiment reported here was undertaken following the indication that the continuous feeding of a ration containing chlortetracycline (Aureomycin)* reduced the incidence of cervical abscesses, increased weight gains, improved feed efficiency, and reduced the amount of time required for pigs to reach market weight.[4]

These beneficial effects were observed in four experiments, using 418 pigs from fall and spring litters, in a herd of pigs on a farm where cervical abscesses had been a recurring problem. The pigs were self-fed either a basal ration or the basal ration supplemented with various levels of chlortetracycline. In all instances, better results were obtained when rations containing chlortetracycline were fed. With 50 Gm. of chlortetracycline per ton of feed, results were better than at lower levels, but there was no apparent advantage in using a higher level under the conditions of the experiment.

The objective of this experiment was to study the preventive effect that continuous feeding of chlortetracycline might exert on the incidence of cervical abscesses in experimentally infected pigs.

## MATERIALS AND METHODS

Twenty-nine pigs weighing 70 to 80 lb., from one farm, were randomly allotted to three groups

From the American Cyanamid Co., Pearl River, N. Y.

The authors thank Mr. Edward Gianforte for the diagnostic work, and Mr. Raymond Adamski for aid in conducting the experiment.

*The trademark of American Cyanamid Co. for the antibiotic, chlortetracycline, is Aureomycin.

of 9 or 10 each, for replicate treatment. The pigs were identified by double ear tags, inoculated with a modified live virus hog cholera vaccine (Rovac†); then held for 18 days for adaptation to the premises and to permit the establishment of immunity to hog cholera. During this period, and for the duration of the experiment, the control group was fed the basal ration only, while the other groups were fed either 20 or 50 Gm. of chlortetracycline with the basal ration. At the end of this adjustment period, 5 ml. of a culture of *beta* hemolytic streptococci that had been isolated from cervical abscesses obtained from a previous field experiment were sprayed into the pharyngeal region of each pig.§

Temperatures were recorded for three preinoculation and ten postinoculation days. Pigs were individually weighed on the day of inoculation and the day before slaughter.

The head and jowls of each pig were examined for the presence of abscesses at slaughter 38 days postinoculation. The contents of the abscesses from the jowls of the affected pigs were examined bacteriologically.

Assay of the complete ration for chlortetracycline content was within the theoretical range.

†The trademark of American Cyanamid Co. for a hog cholera vaccine.

§A Burgess vibrator paint sprayer was used. The culture had been standardized to 40 per cent light transmission on a Lumetron colorimeter using a green filter No. 530.

## RESULTS AND DISCUSSION

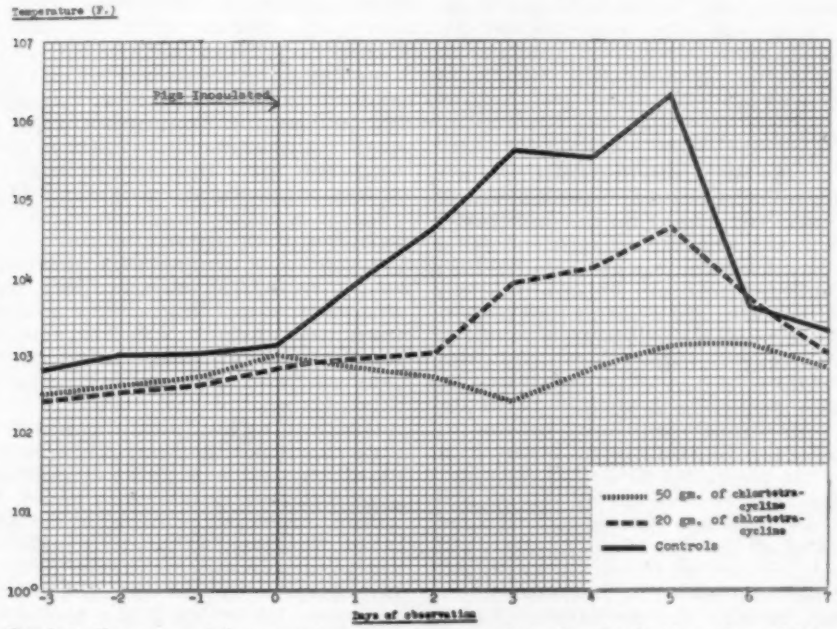
A summary of the over-all results is shown (table 1). The majority of the artificially induced abscesses were deep-seated, measured from 3 to 10 cm. in diameter, and were not observable until after slaughter. While no jowl abscesses were observed in the 10 pigs fed 50 Gm. of chlortetracycline per ton of feed, abscesses were found in 3 of the 9 pigs that received 20 Gm. and in 9 of the 10 control pigs. A species of *beta* hemolytic Streptococcus, the only organism isolated from the contents, was present in all 12 abscesses.

TABLE 1—The Influence of Various Levels of Chlortetracycline on the Prevention of Cervical Abscesses, Weight Gains, and Feed Efficiency of Swine

| Chlortetracycline (Gm./ton)* | 0 | 20 | 50 |
|---|---|---|---|
| No. of animals | 10 | 9 | 10 |
| Heads condemned (abscesses) at slaughter | 9 | 3 | 0 |
| Av. wt. at inoculation (lb.) | 79.4 | 89.11 | 84.30 |
| Av. wt. at slaughter (lb.)** | 153.6 | 158.7 | 153.9 |
| Av. daily gain (lb.) | 1.48 | 1.83 | 1.83 |
| Feed/lb. gain (lb.) | 4.06 | 3.64 | 3.97 |

*Complete ration.

**Thirty-eight days following inoculation with a culture of beta hemolytic streptococci.




Graph 1—The average daily temperatures of three groups of pigs inoculated with a culture of beta hemolytic streptococci.

The average daily temperatures of the pigs in the respective treatment groups are presented graphically (graph 1). An increase in temperature among the control pigs was observed within 24 hours after inoculation and continued until the fifth day when a maximum of 106 F. was reached. After seven days, the temperatures returned to normal. Similar but milder temperature changes were observed in the pigs that received 20 Gm. of chlortetracycline per ton of feed; the maximum average temperature (104.6 F.) also occurring five days after inoculation. There were no observable increases in temperatures in the pigs that were fed 50 Gm. of chlortetracycline per ton of feed.

With the exception of an increase in temperature, no other clinical signs of infection were observed in the two groups of pigs which later developed abscesses.

Weight gains were increased in all antibiotic-fed pigs when compared with the controls. Feed efficiency was lower than observed in earlier experiments, but this may have been influenced by improper functioning of the self-feeders. Nevertheless, feed efficiency was improved in the groups that were fed the antibiotic (table 1). The average daily intake of chlortetracycline in the group fed the higher level of antibiotic was approximately 180 mg., or 1.5 mg. per pound of body weight.

## SUMMARY AND CONCLUSIONS

Twenty-nine pigs were assigned to three replicated experimental treatment groups and were fed a basal ration supplemented with 20 Gm. or 50 Gm. of chlortetracycline per ton. After the pigs had been fed their respective rations for 18 days, each pig was inoculated by spraying the pharyngeal region with a standardized culture of *beta* hemolytic streptococci. All were slaughtered 38 days after inoculation. Abscesses were found in none of the 10 pigs fed the ration containing 50 Gm. of chlortetracycline, but they were present in 3 of the 9 pigs fed 20 Gm. of chlortetracycline, and in 9 of the 10 control pigs. The organism was isolated from the contents of all abscesses. In comparison with the control pigs, an increase in weight gain and an improvement in feed efficiency occurred in those fed the antibiotic-supplemented rations.

The results indicate that the continuous feeding of a ration containing 50 Gm. of chlortetracycline per ton of complete feed will aid in the prevention of cervical abscesses caused by species of *beta* hemolytic streptococci.

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## Conflicting Views on Scrapie

Two views on scrapie and related myopathies in sheep are presented. Ten different syndromes were described when 448 cases of myopathy in sheep from 25 flocks, in Britain, were classified. In four syndromes, rubbing was a prominent feature. These were called: classical scrapie, nonwasting scrapie, asthenic cachetic scrapie, and amaurotic scrapie with progressive loss of vision. The six nonrubbing forms described were: inapparent myopathy (preclinical scrapie), acute hind limb palsey, postpartum pining, acute asthenic paralysis, juvenile pining, and generalized ataxic pining.

Available evidence was considered incompatible with the theory that scrapie is caused by an infectious communicable agent with a long incubation period. The author's hypothesis is that all of the myopathies described, including scrapie, are forms of a disease which is primarily hereditary but subject to modification by environmental factors. It is suggested that control efforts be directed toward eliminating scrapie strains in a breed rather than

by flock elimination.—*H. B. Parry in Vet. Rec., 69, (Jan. 19, 1957): 43-55.*

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Conversely, it is believed that when vacuolated nerve cells are found in the medulla oblongata, in animals which have shown typical signs of scrapie, that this is sufficient evidence for a positive diagnosis. As supporting evidence, it was reported that, when 12 goats were inoculated intracerebrally with brain material from an affected sheep, all of them developed clinical scrapie, as did 8 of 10 goats subinoculated from 1 of the 12. None of 650 other goats which had passed through the flock since 1949 had ever shown signs of a nervous disorder.—*I. H. Pattison in Lancet, 272, (1957): 104-105; abstr. in Vet. Bull. (July, 1957): Items 2183 and 2184.*

## Nocardia Infection of the Bovine Mammary Gland — A Preliminary Report

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This laboratory has repeatedly isolated an organism of the Nocardia group from a single dairy herd in the southern coastal area of California since April 1, 1957.

Of 29 animals, whose milk has been examined bacteriologically on one to three separate occasions, 16 have shed the organism either as a pure culture or in combination with *Micrococcus pyogenes* or *Streptococcus agalactiae*.

The organism appears as gram-positive, branching filaments (fig. 1) in young serum broth cultures, varying to rod and shorter coccoid elements in older cultures. Its growth is aerobic, which distinguishes it from the more familiar pathogens of the Actinomyces group.

The colonies on tryptose agar containing 5 per cent washed bovine erythrocytes appear after 48 hours' incubation. They are nonhemolytic, raised, slightly granular, and whitish. With prolonged incubation (72 to 96 hr.), a yellow-orange pigment develops. No aerial mycelia are formed but the colonies are adherent and appear embedded in the agar. The organism is non-acid-fast.

From the School of Veterinary Medicine, University of California, Davis.

Serum broth cultures, 24 and 72 hours old, have been injected intraperitoneally into guinea pigs and mice. The organism was highly pathogenic to guinea pigs, with death occurring in three to eight days. Raised, white lesions, 1 to 2 mm. in diameter, were evident on the peritoneal surface, from which the organism could be reisolated. Mice were not visibly affected.

Fig. 1—Nocardia sp. isolated from a bovine mammary gland (24-hour broth culture). Gram's stain; x 1,500.

Clinically affected animals have shown extremely fibrous udders. Palpation often reveals areas varying from discrete hard lumps to diffuse fibrosis. Clinical infections have appeared in young, first-lactation heifers and in aged animals.

Clinical signs vary from local inflammation in the udder, accompanied by severe C.M.T. reactions[1] and, in some instances, by frank clots of pus in the milk, to visible malaise, anorexia, and febrile reaction.

The udder of an animal from which the organism was isolated in pure culture on

[1]Schalm, O. W., and Noorlander, D. O.: The California Mastitis Test. California Vet., 9, (1956):33.

three consecutive occasions was observed at slaughter. The cut surface of the udder showed several thick-walled lesions, 2.5 to 5.0 cm. in diameter, filled with a pasty granular pus. Other areas were more diffusely fibrous. The organism was recovered from various areas of this gland.

To date, the infection has proved highly refractory to both chemotherapeutic and antibiotic agents.

Studies are in progress to establish, if possible, positive identification of the causative organism, pathogenesis, and pathological findings in both naturally and experimentally infected animals.

## Effect of *Leptospira Pomona* Bacterin upon Agglutinin Titers of Five Cattle Previously Vaccinated with Strain 19 *Brucella Abortus*

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Since the practical diagnosis of brucellosis in cattle is based upon the agglutination test, it is of the utmost importance to determine whether an injection of a given biological product, other than Brucella vaccine, or infection with an etiological agent other than Brucella, may stimulate the production of agglutinins which could react with Brucella antigen. Recently, it was reported that injection of Pasteurella bacterin into cattle vaccinated with strain 19 *Brucella abortus* as calves may give rise to an anamnestic reaction.[1,2] In such instances, a temporary reappearance of agglutinins to Brucella antigen was observed in animals which had lost their Brucella titers, or a rise in a declining titer was noticed.

To determine whether the injection of *Leptospira pomona* bacterin might have an anamnestic effect upon animals previously injected with strain 19 of *Br. abortus*, a preliminary study was carried out on 5 animals, 2 Guernsey cows and 3 Holstein-Friesian heifers. Each of these had been vaccinated with strain 19 *Br. abortus* ten to 34 months previously, when they were between 6 and 8 months of age. When injected subcutaneously with 5 ml. of *L. pomona* bacterin,* their Brucella agglutinin titers ranged from incomplete in 1:20 dilution to incomplete in 1:80 dilution of the serum. Blood samples were obtained from all 5 animals one, four, and ten weeks after injection of the Leptospira bacterin, and the serums were tested for the concentration of Brucella agglutinins beginning with a 1:10 dilution of serum (table 1). There was no detectable rise in the agglutinin titers during this period when an anamnestic reaction would have occurred.

TABLE 1—Results of Injection of Leptospira Pomona Bacterin Upon Agglutinin Titers of Cattle Previously Vaccinated with Strain 19 Brucella Abortus

| Animal No. | Interval between injection of Brucella vaccine and Leptospira vaccine | Agglutination titer | | | |
|---|---|---|---|---|---|
| | | At time of injection of Leptospira vaccine | 1 week later | 4 weeks later | 10 weeks later |
| G 1 | 34 mo. | P 1:20* | P 1:20 | P 1:20 | P 1:20 |
| G 2 | 23 mo. | +1:20 | P 1:20 | +1:20 | P 1:20 |
| H 1 | 10 mo. | P 1:40 | P 1:20 | P 1:20 | P 1:40 |
| H 2 | 11 mo. | P 1:40 | P 1:40 | P 1:40 | P 1:40 |
| H 3 | 12 mo. | P 1:80 | P 1:40 | P 1:40 | P 1:40 |

*+ = complete agglutination; P = incomplete agglutination.

### SUMMARY

In this limited study, there was no indication of a rise in Brucella agglutinins in the blood serum of 5 cattle which had been vaccinated with strain 19 *Brucella abortus* during calfhood, and injected with *Leptospira pomona* bacterin ten to 34 months later. Although the number of animals used was small, the results are presented since, as far as is known, such studies have not previously been reported.

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*Antilepto, Merck Sharp & Dohme.

# Ringworm in Horses Caused by the Dermatophyte, *Microsporum Gypseum*

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IN A PREVIOUS report,[1] the clinical and mycological findings in a horse spontaneously infected by the dermatophyte, *Microsporum gypseum* (*M. fulvum, Achorion gypseum*), were described. This was the first such equine infection to be recorded from the United States. The common occurrence of *M. gypseum* in soil, and the apparent ample opportunity for horses to come in contact with this fungus suggested the possibility that cultural studies might show this dermatophyte to be an important cause of ringworm in horses in the United States.

This paper reports the first epizootic, of *M. gypseum* ringworm in horses in this country. This agent has been previously implicated in an epizootic of ringworm in a Belgian remount station.[2]

## CLINICAL HISTORY

An Atlanta, Ga., resident owned 11 horses, kept on a 25-acre farm located in the suburbs, consisting of 6 Shetland ponies, 1 Welsh pony, and 4 riding horses of mixed breed (table 1). In addition, the owner kept a male Boxer dog 2 years old, and 3 cats on the premises. Periodically, the horses were brought in from pasture for grooming, saddling, and supplemental feeding and, during inclement weather, they were kept in the barn.

On Oct. 6, 1956, a number of raised, circular areas were noticed on the back and shoulders of 1 of the animals. Within a few days, 7 other horses showed similar elevations on various parts of their bodies. It was thought the lesions were caused by insect bites. Eighteen days later, one of the authors (J.L.H.) was called to treat the horses for intestinal parasites and to determine the cause of the cutaneous abnormalities. Since the lesions resembled ringworm, hair and skin scrapings from 1 of the animals were submitted to a laboratory* for mycological examination. The affected areas were clipped and a topical application of a medication containing flowers of sulfur, oil of turpentine, oil of tar, and raw linseed oil was prescribed. The owners were cautioned to exercise care to avoid human infections. At the laboratory, the direct microscopic examination of the material showed both hair and skin invasion by a dermatophyte. A presumptive laboratory diagnosis of ringworm was made.

On October 28, an investigation was initiated. All animals on the farm, except 2 cats, were carefully examined, and materials for laboratory study were collected from each. Hairs and crusts or scales were collected by plucking or scraping affected areas with sterile forceps, a separate pair being employed for each animal. A few hairs were taken from the head, back, and tail regions of the apparently normal animals. Of the 11 horses, 8 showed multiple circular crusted areas, with the hair protruding through the crusts. In some of the areas, the hairs appeared to be matted by dried exudate (fig. 1). Many of the lesions were not readily visible but could be easily felt. The individual lesions varied in size from a few to 30 mm. and were distributed over the entire body of several affected horses (table 1). The hairs were difficult to epilate and when forcibly removed, together with the adherent crusts, reddened, moist areas of alopecia remained. The lesions did not appear to cause any discomfort to the horses.

The Boxer dog showed one circular, scaly area of alopecia (1.7 cm. diam.) on the left side of the neck. The cat that was examined did not show visible cutaneous abnormalities.

A total of 12 soil samples for laboratory study was collected from within the barn, from its immediate vicinity, and from the pasture, by scraping the soil directly into 4-oz. jars.

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*Mycology laboratory of the Communicable Disease Center, Chamblee, Ga.

TABLE I—Dermatophytes Recovered and Location of Lesions in Horses Studied

| Identification No. | Age | Sex | Breed | Locations of Lesions | Dermatophyte isolated |
|---|---|---|---|---|---|
| 1 | 6 mo. | M | Shetland | Multiple, entire body. | M. gypseum |
| 2 | 15 mo. | M | Shetland | Multiple, entire body. | M. gypseum |
| 3 | 4 yr. | M | Shetland | None. | None |
| 4 | 8 yr. | F | Shetland | Multiple, flank, and back. | M. gypseum |
| 5 | 9 yr. | F | Shetland | None. | None |
| 6 | 12 yr. | F | Shetland | Multiple, head, and neck. | M. gypseum |
| 7 | 9 yr. | F | Welsh | Multiple, shoulder, back, flank. | M. gypseum |
| 8 | 4 mo. | F | Saddle | Multiple, entire body. | M. gypseum |
| 9 | 6 mo. | F | Saddle | Multiple, entire body. | M. gypseum |
| 10 | 5 yr. | F | Saddle | Multiple, entire body. | M. gypseum |
| 11 | 8 yr. | F | Saddle | None. | None |

The premises were revisited at one- or two-week intervals to observe the progress of the disease. By Nov. 10, 1956, some of the horses showed bare circular areas where the crusts had fallen off. Eight days later, all the affected horses showed numerous circular, scaly areas of alopecia and the flanks, sides, and other sites on some presented a "moth-eaten" appearance, with loss or breakage of hairs (fig. 2). The lesions on the Boxer showed evidence of healing and regrowth of hair.

Eight weeks after first noticed, the areas of alopecia were still visible on the horses but the hair was returning and all the crusts had fallen off. The lesion on the Boxer had completely healed, without scar formation. By the twelfth week, the lesions on the horses had completely healed without scar formation and no new lesions had developed.

Although the five members of the owner's family (ages 4 to 40 years) had repeated contacts with the infected horses and the

Fig. 1—Early ringworm lesions, on the pastern region of a horse, appearing as raised circular crusted areas, with hairs protruding through the crusts (a). Early lesions, appearing as raised crusted areas, on the neck and shoulder region of a horse—the hairs appear to be matted by dried exudate (b).

dog, there was no evidence of transmission of the infection to them.

## MYCOLOGICAL FINDINGS

The direct microscopic examination of the clinical materials showed evidence of a dermatophyte invasion in the 8 horses and the Boxer. This procedure consisted of placing a few hairs and skin scrapings in a drop of 10 per cent potassium hydroxide on a glass slide, adding a cover slip, and gently heating the preparation over a Bunsen burner for a few seconds to hasten clearing. When examined under low and high power of the microscope, an ectothrix pattern of hair invasion was observed. Large spores (5 to 8 $\mu$) were seen in chains and irregular masses on the surface of the hairs, and mycelial filaments were observed within the hairs. In the skin scrapings, branching, septate mycelia with arthrospore formation were readily visible. In no instance was the characteristic Microsporum-type hair invasion, as seen in *M. canis* infections (sheath of small spores, 2 to 3 $\mu$ in diameter, in mosiac), observed. None of the specimens fluoresced when examined under the Wood's light.

To isolate and identify the dermatophyte involved, all specimens were cultured on a special selective-isolation medium, containing the antibiotics cycloheximide, penicillin, and streptomycin,[4] and incubated at room temperature (25 C.).

The materials collected from the 8 horses and the dog yielded, in five days, a fungus which was identified on the basis of its gross and microscopic characteristics as *M. gypseum*. Macroscopically, the colonies were flat, powdery, and cinnamon brown, with a tannish pigment on the reverse side. The direct microscopic examination of the colonies showed the presence of numerous elliptical multiseptate, rough-walled macroconidia. A few microconidia were observed in some of the isolates.

The soil specimens collected were tested for the presence of keratinophilic fungi by employing the

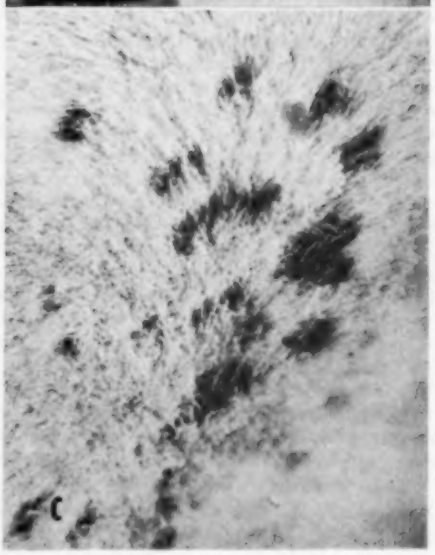


Fig. 2 (above and right)—Late lesions, appearing as circular scaly areas of alopecia, on the face of a young pony (a). Late lesion, appearing as circular scaly area of alopecia, on fetlock of a horse (b, upper right). Close-up of flank region of a horse, presenting a "moth-eaten" appearance (c, lower right).

"hair bait" technique.[5] The dermatophyte, *M. gypseum*, was recovered from seven of nine samples gathered from the vicinity of the barn and from the pasture. Three samples collected from inside the barn did not yield this fungus.

## DISCUSSION

Epizootics and isolated cases of ringworm in horses have been reported previously from this country.[5-7] Where cultural studies supplemented clinical diagnoses, the etiological agents described have been various members of the genus Trichophyton, including *T. equinum*, *T. mentagrophytes*, *T. verrucosum*, and the organism *M. canis* (*M. equinum*). These dermatophytes are, as far as is known, parasitic in habit. They are readily transmitted from one host to another and may cause infections of large numbers of susceptible animals. On the other hand, *Microsporum gypseum* is essentially a soil saprophyte and does not appear to require tissues of living animals for propagation. Due to its widespread distribution in our environment, man and lower animals are constantly exposed to this fungus. The potential hosts usually appear to resist invasion but sometimes the fungus does penetrate the defense mechanisms to produce infections. The full explanation for such invasions is not known but, under certain conditions, some strains of *M. gypseum* may possess increased virulence or the resistance of the host tissues may be reduced at the time of contact with the organism.

In this epizootic, neither the age nor the type of the horses seemed to affect the susceptibility of the animal. However, the horses did have one thing in common—all were heavily parasitized by intestinal worms and were in poor condition, which probably was an important factor in permitting cutaneous invasion by the dermatophyte. The fact that practically all the affected horses showed evidence of disease within one week suggests a simultaneous exposure and, since the organism was readily demonstrated in soil samples, it is possible that direct contact with such soil was the source. It is also possible that the infection developed in one animal and the fungus was transferred from it to the others through the medium of contaminated grooming tools or other equipment.

The fact that similar conditions, *i.e.*, horses in poor condition due to intestinal parasites or other causes, and the presence of the agent in the environment, can be found in most parts of the country, suggests the distinct possibility that cultural studies of equine dermatophytoses may show *M. gypseum* to be an important cause of ringworm in the United States. Since the clinical picture of this type of ringworm is essentially the same as infections caused by other dermatophytes, cultural studies would be necessary to confirm this.

## SUMMARY

The clinical and mycological findings in ringworm in horses, caused by *Microsporum gypseum*, are described. The only dog and 8 of 11 horses on the premises developed clinical cases of ringworm. There was no evidence of transmission of infection to human contacts. This is the first such authenticated epizootic to be reported from the United States.

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*Antifoam Agents in Pulmonary Edema.*—Frothy edema in the alveoli hinders the diffusion of oxygen which sets up a vicious circle since the adjacent respiratory epithelium then becomes anoxic and more permeable to fluid. When antifoam agents were tested on rabbits with induced pulmonary edema, a mixture of a silicon and a polyhydric alcohol compared favorably with 95 per cent ethyl alcohol. When nebulized and given to eight patients, by inhalation with an intermittent positive-pressure unit, the results were excellent. —*J.Am.M.A. (Feb. 23, 1957): 630.*

# Oxygen Therapy with a Nasopharyngeal Tube in Large Animals

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OXYGEN THERAPY has not been fully developed in veterinary medicine even though it offers great potential benefit. It was first applied in small animal practice[6,7] and, more recently on a limited scale, in horses and cattle.[9,10]

The development of the sulfonamides and antibiotics, with their outstanding contribution to the improvement of therapy, has probably diverted attention from a more extensive use of oxygen therapy. However, in certain disorders such as pneumonia these drugs can arrest infection, but inflammatory exudates must be absorbed. All too often this absorption is slow and incomplete. The function of the diseased portion of the lungs must be taken over by the healthy lung tissue. This is often done successfully, but occasionally the lungs can not cope with the disease and emphysema or recurrence of pneumonia may result, terminating in death. In such instances, oxygen therapy can often save the life of the animal.

Another disorder in which oxygen therapy is logical, and often the only one of value, is acute pulmonary emphysema, sometimes encountered in cattle in the spring and fall. Its cause is obscure; possibly some kind of allergy. Treatment is seldom effective since the animal usually dies in a few hours, but oxygen with antiallergic agents is indicated.

## OXYGEN PHYSIOTHERAPY

Oxygen accounts for approximately one fifth of the total atmosphere. This is normally sufficient for respiration, provided the transport and utilization of oxygen are not impaired. A smaller quantity of oxygen in the blood and body cells, regardless of the cause, constitutes hypoxemia, of which there are several types:

*Hypoxic Hypoxemia.*—This type of hypoxemia occurs when insufficient amounts of air oxygen reach the blood circulating through the lungs. All pathological conditions resulting in insufficient lung ventilation fall into this category: pneumonias, lung edema, acute and chronic lung emphysema, or obstruction or constriction of any part of the air passage to the lungs by foreign bodies, pathological processes, or excessive mucus.

*Anemic Hypoxemia.*—This hypoxemia is encountered in all conditions associated with a decrease in hemoglobin, such as the primary and secondary anemias.

*Stagnant Hypoxemia.*—Stagnant hypoxemia results from insufficient circulation of the blood, even though its oxygen and hemoglobin content are normal. This hypoxemia occurs in heart failure, pericarditis, circulatory collapse, and shock—both postoperative and anaphylactic.

*Histotoxic Hypoxemia.*—This type of hypoxemia occurs when the normal amount of oxygen in the blood reaches the cells but the cells are not able to utilize it, a rare condition encountered in cyanide poisonings and, to some extent, during general anesthesia.

*Mechanism of Respiration.*—The mechanism of respiration is under the control of the respiratory center in the medulla oblongata. The activity of this center is regulated by carbon dioxide in the blood stream as well as by carbon dioxide produced by this center itself. Reflexes also play an important part. Lack of oxygen is the most important depressor of the respiratory center.

It has been shown that there is an increase in permeability of the lung capillaries when oxygen is lacking.[3] Plasma passes through the capillary walls and separates the alveolar epithelium from the endothelium of the capillaries. As the transudation progresses, pulmonary edema develops and the capillaries are pushed away from the oxygen of the alveoli. This aggravates the hypoxemia and, subsequently, leakage of the lung capillaries becomes more extensive, developing a vicious circle.

From the Department of Veterinary Science, University of Wisconsin, Madison.

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The author thanks Mr. C. E. Zehner, assistant professor in the Department of Dairy Husbandry, University of Wisconsin, for his assistance and suggestions.

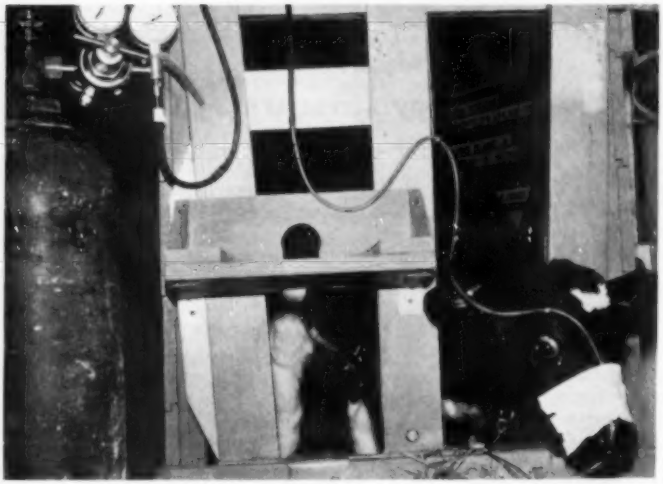

Fig. 1—A calf, 3 days old, receiving oxygen therapy.

Finally, hypoxemia can reach a stage that is not reversible unless pure oxygen is administered. A similar situation develops in all pneumonic conditions when edematous fluid with inflammatory exudate accumulates around the capillaries, in interstitial tissue, and the alveoli.

The therapeutic value of pure oxygen has been demonstrated.[2,3,5,11,12] It restores the normal oxygen content of hemoglobin, through the unaffected portion of the lungs, and also the still functioning, though diseased, parts of the lungs. Pure oxygen possesses five times more power to penetrate fluid than does the oxygen in atmospheric air. Hemoglobin is the principal carrier of oxygen from the lungs to the tissues. Normally, in man, each 100 ml. of blood which leaves the lungs contains 20 ml. of oxygen in the form of oxyhemoglobin. Tissues utilize only about 5 ml. of this oxygen; thus 15 ml. is returned to the lungs.

Blood plasma normally carries only a small amount of oxygen. However, when the oxygen concentration of inspired air is changed from its normal 20.9 per cent to 35.0 to 50.0 per cent, the plasma can carry 1 ml. of oxygen per 100 ml. of plasma. Since this is available to the tissues, it means a 16.6 per cent increase in available oxygen. Also, the dissociation of oxygen from the oxyhemoglobin takes place faster when the blood is more nearly saturated with oxygen.

## MATERIALS AND METHODS

An oxygen mask for large animals[10] and a homemade oxygen tent[9] have been described, but simpler, more practical, and less expensive equipment is needed.

The equipment selected includes 7 to 8 ft. of flexible plastic tubing, ⅛ to ¼ inch in diameter; a two-stage regulator with a flow gauge; and a cylinder of oxygen. The regulator has two indicators; one for measuring the flow of oxygen per minute, the second for measuring the cylinder's pressure and contents. The tube should be lubricated and inserted into the nasal passage to a depth equal to the distance between the nostril and a point one third of the distance between the lateral canthus of the eye and the base of the ear. If the animal shows the swallowing reflex, the tube should be pulled back until swallowing ceases. The tube is fixed with adhesive tape to the external edge of the nasal opening, or to the nose ring in bulls, then is turned back and fastened to the head or halter, then to the stanchion, and connected to the cylinder (fig. 1). It is important that the tube be inserted to the correct depth, otherwise much of the oxygen will be wasted. At least once a day, the tube should be removed, cleaned, disinfected, and inserted in the opposite nostril.

For the first several hours of oxygen administration, the flow should be rapid. Later, it can be slower. The rate and duration of oxygen administration must be regulated according to the seriousness of the disease and the size of the animal. A flow of 4, 6, and 8 liters per minute will give approximately 35, 45, or 50 per cent respectively, of bronchial oxygen concentration in animals weighing ±200 pounds.[1] Usually, the more severe the hypoxemia, the more dramatic is the improvement following oxygen therapy.

An additional unit, the nebulizer, can be attached and will assure proper humidification of

the oxygen to prevent drying of the mucous membranes, as well as to administer aerosols of antibiotics,[10] antihistamines, bronchiodilators, or other chemotherapeutic agents. When using an aerosol nebulizer, it must be two thirds full and fine bubbles of oxygen must be passing through it.

Commercial oxygen, as used by welders, is satisfactory for animals and can be purchased in economical large cylinders (medical oxygen is not required). A small cylinder can be kept in the car at all times for an emergency.

## CASE REPORTS

*Case* 1.—On Dec. 6, 1956, a Jersey heifer, 8 weeks old, was treated for acute bronchopneumonia with penicillin, streptomycin, and triple sulfonamides for five days. Her body temperature dropped from 106.8 to 103.0 F. on the third day of treatment, but the rate of respiration continued at 150 to 160 per minute. During the next three days, the type of respiration became more alarming in spite of antibiotic and glucose infusion therapy. Moist bronchial râles and crepitations were audible on both sides over the ventro-posterior and central areas of the chest wall. The calf stood with its front legs spread, head lowered, neck extended, and tongue protruded. Pronounced dyspnea was manifested by mouth breathing, accumulation of froth around the lower lip, dilation of nostrils, collapse of the intercostal spaces, abdominal breathing, and slight to-and-fro movements of the whole body. Visible mucous membranes were cyanotic, the heart rate was 115 to 120 per minute, and the appetite ceased.

As a last resort, oxygen therapy was applied. A flow of 5 to 6 liters per minute was given day and night for two days. After 24 hours, breathing became deeper, the rate slowed to 80 per minute, the calf started to drink milk, and it lay down for the first time in several days. After 48 hours of oxygen therapy, the heart and respiration rates and body temperature were within normal limits. Crepitation and fine râles were still audible although the calf breathed comfortably in normal atmosphere. Oxygen therapy was continued for two more days (not nights) with a flow of 3 liters per hour to allow more complete absorption of the exudate. Recovery followed.

*Case* 2.—On Feb. 24, 1957, another calf (fig. 1), 3 days old, which had shown slight dyspnea since birth, probably due to inhalation of mucus, developed an almost identical bronchopneumonia. When the sulfonamide-antibiotic therapy did not bring satisfactory results, oxygen therapy again resulted in a prompt recovery.

*Case* 3.—On March 22, 1957, a Holstein-Friesian bull, 7 years old, suddenly developed inspiratory and expiratory dyspnea with loud inspiratory snoring, as a result of stenosis of the upper respiratory passage. The temperature was 102.8 F., the respiration rate 22, and the heart rate 58 per minute. A standard sized stomach tube was too large for nasal passage so a tube ¼ inch in diameter was used. Oxygen was given daily, 7 liters per minute in the morning and 4 liters per minute in the afternoon. Antibiotics were also given. The snoring gradually diminished and after four days the respiration and temperature were normal. The bull recovered.

## SUMMARY

The physiotherapy of oxygen is reviewed. Simple, economical, and efficient equipment for nasopharyngeal administration of oxygen in large animals is described. Its successful use in 3 animals is reported.

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# Pulmonary Vascular Sclerosis as a Result of *Dirofilaria Immitis* Infection in Dogs

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SOME YEARS AGO, several local veterinarians afforded us the opportunity to study the hearts and lungs of dogs suffering from cardiopulmonary disease of the nature of an endarteritis obliterans of the pulmonary arterial tree. These dogs, usually 1 year or older, had obvious shortness of breath and resisted forced exertion. At necropsy, there was evidence of cor pulmonale and morphological reflections in the arterial vessels of hypertension. Adult forms of the filarial worm *Dirofilaria immitis* were found in the right side of the heart and in the pulmonary arterial system. Thus, a natural disease in the dog afforded us an opportunity to study obstructive vascular disease of the lungs and its effect upon the heart.

Fig. 1—Transverse section of a dog's heart showing left and right ventricular hypertrophy. Arrow points to the right ventricle.

Fig. 2—Adult worms in the lumen of the right ventricle of a dog's heart.

This initial study comprised a careful gross and microscopic examination of dogs with obvious clinical disease manifested by shortness of breath, ascites, and slowness on exertion. These dogs were from the tidewater area of Virginia where the filarial worm is endemic. A group of dogs with latent filariasis, selected by veterinarians who made blood smears and discovered the filarial forms "swimming between the blood cells," was also studied.

From the Department of Pathology, Medical College of Virginia, Richmond. Dr. Hennigar is associate professor of pathology and Dr. Ferguson is an associate in medicine and research fellow in pathology at the Medical College of Virginia.

The authors thank Dr. Averill Liebow, professor of pathology, Yale University, for his advice, and Melvin C. Shaffer, director of visual education, Medical College of Virginia, for preparation of the photographs.

These studies were supported by grant HE-2184 from the Division of Research Grants, National Institutes of Health, Education, and Welfare, Bethesda, Md.

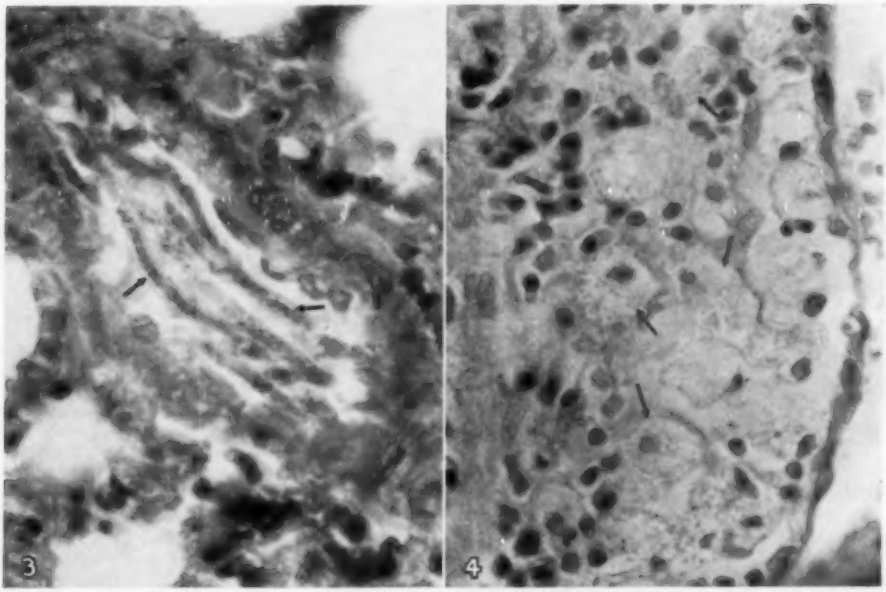

Fig. 3—Microfilaria (arrows) in a small pulmonary artery of a dog. Masson's trichrome stain; x 374.
Fig. 4—Large pulmonary artery of a dog, showing atherosclerosis. Numerous lipophages (arrows) in the intima are accompanied by plasma cells and lymphocytes. H & E stain; x 374.

## NECROPSY FINDINGS

On necropsy of the first group showing clinical disease, the entire right side of the heart was found to be markedly dilated and hypertrophied, and there was occasional hypertrophy on the left side (fig. 1). Masses of adult worms were found in the right ventricle and right atrium (fig. 2). More often than not, numerous adult worms could be traced into the pulmonary arterial tree for several centimeters, even to the point of causing partial mechanical obstruction. Adult worms were never found in the left side of the heart.

Only filarial forms were found in the small arteries and arterioles (fig. 3). On the venous side, filarial forms but no adult parasites were demonstrated in the lumen of the vessels. Histological examination of the larger pulmonary arteries revealed atherosclerosis (fig. 4) and endothelial cushions (fig. 5). Focal intimal hemorrhage was occasionally seen in these larger vessels. The smaller arteries showed medial hypertrophy and endothelial thickening (fig. 6), while the arterioles were characterized primarily by medial hypertrophy (fig. 7). No corresponding medial or in-

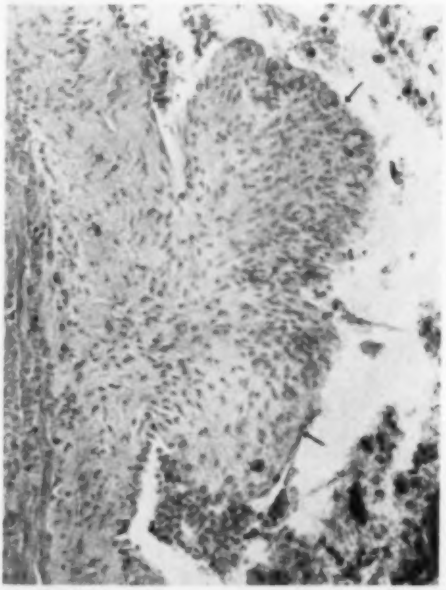

Fig. 5—Large pulmonary artery of a dog showing fibrous endothelial cushion (arrows); calcified thrombus material in adjacent lumen (black). H & E stain; x 251.

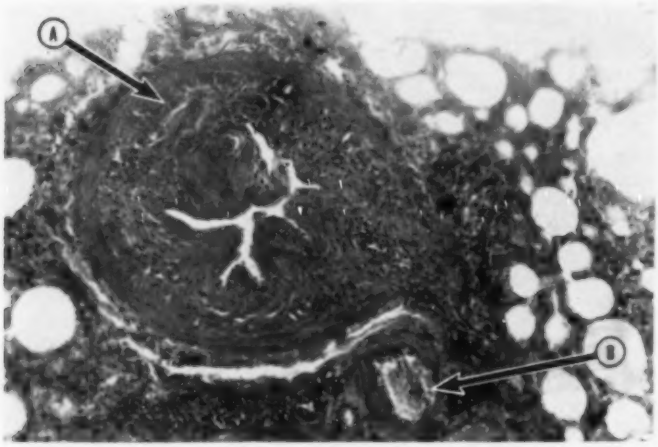


Fig. 6—Intimal fibrosis of a small pulmonary artery (A) and prominent bronchial artery (B) in a dog. H & E stain; x 133.

timal lesions were found on the venous side, but often the veins and venules were dilated and around these were extravasation of red cells and fibrin-rich exudate (fig. 8). Occasionally, there were perivascular hemorrhages and frequently the bronchial vessels in the walls of bronchi and bronchioles were dilated and prominent. Except in some of the older dogs in which emphysema was found, there were no parenchymal changes. Passive congestion of the other organs was also found.

In the second group of dogs, those with subclinical or latent signs, minor degrees of the above findings were observed.

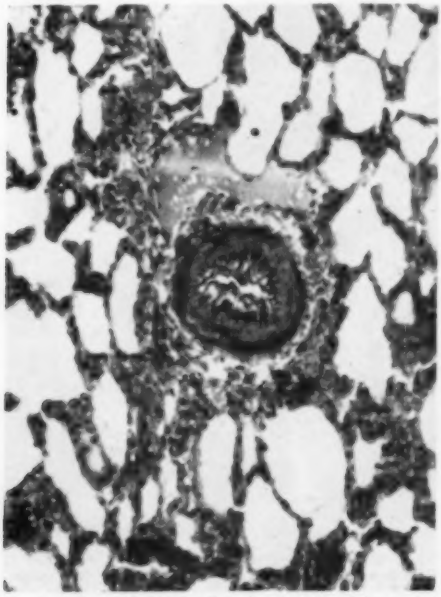

Fig. 7—Medial hypertrophy of arterioles. Masson trichrome stain; x 200.

Fig. 8—A dog's vein with hemorrhagic extravasation. H & E stain; x 67.

The dogs with clinical manifestations and those without signs of infection, but in whom microfilaria were demonstrated in the blood, were studied with respect to cardiac output and pressures in the right side of the heart and pulmonary arterial tree. These studies were carried out by one of us (R.W.F.) with cooperation of the cardiovascular group* at the Medical College of Virginia. The results of these circulatory dynamic findings will be reported.[7]

## THE PARASITE

The morphological characteristics of the microfilaria are similar to the microfilaria of *Wuchereria bancrofti* (fig. 9) in man. The adult worm has been morphologically described by numerous investigators (fig. 10).[1,2] The mode of transmission of the parasite of heartworm disease of the dog is far from clear. It is believed that the female mosquito is the vector in the lowland part of the tidewater area of Virginia.[6] We have attempted to transmit the disease by direct blood transfusions in 3 normal dogs but this has not proved successful. Further effort should be attempted along this line. Adult filarial worms have been found outside the vascular tree in dogs.[6] Adult and young worms collected directly from the heart by dissection and inserted surgically into the subcutaneous tissue of a dog (free from heartworm infection) were later found in the heart.[4]

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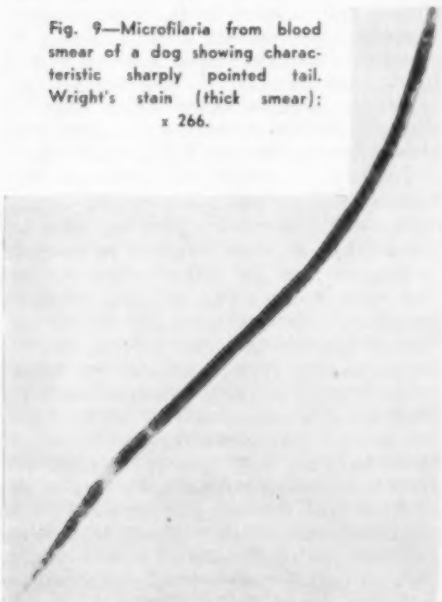

Fig. 9—Microfilaria from blood smear of a dog showing characteristic sharply pointed tail. Wright's stain (thick smear); x 266.

## DISCUSSION

It appears to us that it takes nine to 18 months for the development of sufficient pulmonary arterial damage before right ventricular strain becomes manifest.

The nature of the endothelial injury caused by the adult worms or larvae is not clearly understood. The possibility of its being a hypersensitivity inflammatory reaction is in need of investigation. On the other hand, the mechanical irritation may be all important, particularly in the large vessels.

Few experimental conditions have been established which have successfully lent themselves to the study of chronic pulmonary hypertension and cor pulmonale, although some notable work in this di-

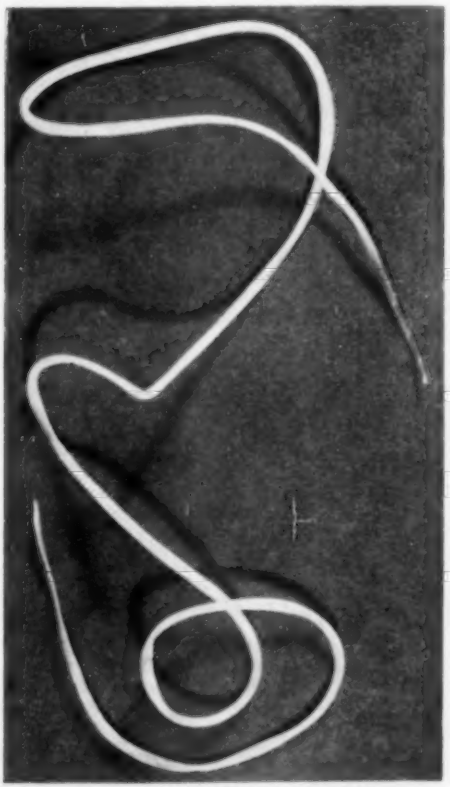

Fig.—10 Adult Dirofilaria immitis. x 1.2.

rection has been reported.[2,5] Recently, the effects of the introduction of plastic beads into the pulmonary arteries of rabbits have been studied[8] experimentally.

It would appear that a study of dogs might shed some light on such matters as the length of time required to develop cor pulmonale and the extent to which this may occur.

In addition, the study of the anastomoses between the pulmonary and bronchial arteries, as well as the venous return of the lungs, may be revealing as to the direction and extent of the blood flow through the lungs under the conditions of pulmonary vascular obstruction.

We have made a number of vinylite plastic casts of the lungs of dogs with filarial pulmonary endarteritis obliterans in order to study the circulation and this will be the subject of a separate report.[7]

## SUMMARY

1) Dogs with clinical and subclinical pulmonary hypertension were studied from the standpoint of morphological findings.

2) Endarteritis obliterans of the pulmonary arteries due to infection with *Dirofilaria immitis* is demonstrated.

3) The possibilities of this condition in the dog as an experimental tool in the study of chronic pulmonary hypertension and cor pulmonale are emphasized.

4) Circulatory dynamic studies on animals such as the ones described may assist in an understanding of the pathophysiology of the circulation in pulmonary vascular obstruction.

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*Brucella Melitensis in Goat Milk.*—When 58 milking goats were experimentally infected with *Br. melitensis*, the organism was recovered from the milk of 34 but not until three weeks after infection. It was not found in any milk sample which was negative to the stained antigen test. It was more frequently found in centrifuged sediment than in the cream.—*Vet. Bull (June, 1957): Item 1696.*

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*No Foci of Strain 19 Brucella in Vaccinal Reactors.*—Tissues from 10 organs of each of 6 cattle which had persistent postvaccinal titers of 1:40 to 1:80, but were negative to milk agglutination tests, were inoculated into guinea pigs and on potato agar plates. Brucella organisms were not isolated or recovered.—*Monatsh. f. Tierhlkd., 8, (1956): 235 (abstr. in German Sci. Bull. (June, 1957): 8).*

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*Brucella in Fetal Membranes.*—When fetal membranes from cows of both vaccinated and infected herds were examined, Brucella were isolated from 7 of the 98 vaccinated animals, only 2 of which showed titers; and from 7 of 39 from infected herds. No Brucella were isolated from the clinically normal membranes of cows with blood titers as high as 1:5,120.—*Monatsh. f. Tierhlkd., 8, (1956): 257 (abstr. in German Sci. Bull. (1956): 8).*

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*Influence of Other Infection on Tetanus.*—Tetanus does not follow every inoculation with spores. The synergic effect of associated infective organisms on the toxicity of *Clostridium tetani* depends on their biological properties. Toxic coliform organisms favor infection, whereas nontoxic coliforms have an inhibitory effect. *Bacillus subtilis* can prevent development of tetanus. Tissue damage, even some distance from the site of infection, favors the development of disease. The development of *Cl. tetani* septicemia, with its shorter incubation period and more violent course, depends on the associated organisms.—*Vet. Bull. (July, 1957): Item 2015.*

## Nutrition and Egg Quality

The amino acid composition of egg protein is quite constant. Significant increases of cystine and methionine is shown when a change is made from a low protein to a high protein ration. Low-fat feeds increase the fat in the egg yolk. Feeds containing large amounts of hemp seed, linseed, and cottonseed oils, as well as fish oils, increase the unsaturated fatty acids in the egg yolks. Deterioration and liquification of egg albumen in storage eggs may be due to the unsaturated fatty acids of the egg yolks acting as reducing agents.

The content in eggs of vitamins A, D, E, K, thiamin, riboflavin, and pantothenic acid may be readily increased by enriching the feeds. This increase in vitamin content is limited because the vitamin transfer from feed to eggs decreases rapidly when the vitamin content of the ration is greatly increased. Feeds high in iodine, bromine, fluorine, manganese, and selenium increase the egg content of these elements.

High humidity and high temperatures decrease the blood calcium levels and the eggshell strength is lowered. For producing strong eggshells, high-grade limestone was equal to the more soluble calcium compounds such as calcium gluconate, calcium lactate, and precipitated chalk.

The number of blood spots in eggs is reduced when hens are on range and when they receive adequate vitamin A. Vitamin K deficiency does not seem to be a cause of blood spots. Eggs produced from rations made with meat scraps were heavier than when the feed was made with soybean meal. Rations high in corn (energy) had no effect on egg weight, but resulted in increased yolk color and a consequent lowering of the candled grade of the eggs. —*Werner J. Mueller in Feed Age, Jan., 1957.*

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*Dairy Calf Feeding Practices* (as reported by dairymen).—(1) About 52 per cent of the calves included in this report were fed whole milk and no grain; (2) 36 per cent were fed whole milk plus a calf starter supplement; (3) 10 per cent were fed a milk substitute mixed with water; (4) the remaining 2 per cent were fed miscellaneous feeds such as skimmed milk, buttermilk, and condensery drippings.

Most dairymen reported that calves grew faster on whole milk.

Where a milk substitute was used, the farmers said the calves had less scours. Those feeding milk substitutes weaned their calves from whole milk at an average of 12 days of age. The substitute was fed until the calves were an average of 76 days old. Many farmers reported feeding the regular dairy ration to their calves. —*Wisconsin Dairying, Jan., 1957.*

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*High Roughage Feeding for Raising Low Cost Dairy Cattle.*—This system of feeding involves: (1) limited milk feeding to calves until they are about 50 days old; (2) free-choice feeding of good quality, mostly legume, hay; (3) the feeding of a grain mixture, roughly about one half the amount of hay being consumed.

Calves were allowed to nurse their dams for the first three days. The amount of whole milk fed to calves varied from 10 to 16 lb. daily. This was gradually reduced to zero at 50 days of age. The suggested grain mixture was: corn 500 lb., oats 365 lb., soybean meal 125 lb., and iodized salt 10 lb. Salt and bone meal were fed free-choice and vitamin A was added to the milk in the late winter and early spring. Growth was satisfactory when the hay and grain were pelleted. Cud inoculations did not improve growth unless the hay was of poor quality.—*Hibbs, Conrad, Pounden in Feed Age, Feb., 1957*

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*Feeding Enzymes to Baby Pigs.*—The effect of supplemental proteolytic enzymes on the digestibility of fish meal and soybean proteins was tested, in Canada, on 31 newborn pigs. Feeding enzymes had no effect on the growth rate or on the digestibility of either plant or animal proteins. When soybean protein which had been predigested for 24 hours with pepsin was fed to pigs 2 days old, all 8 pigs developed a severe diarrhea and died within seven days.—*J. Anim. Sci. (Aug., 1957): 568.*

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*Feeding Yeast to Steers.*—The addition of live-cell yeast to a low-quality roughage, a high-quality roughage, and a fattening-type ration, at the Oklahoma Agricultural Experiment Station, depressed the digestibility of ether extract material in each feed but did not affect the digestibility of other nutrients.—*J. Anim. Sci. (Aug., 1957): 671.*

# Editorial

## Preparing Manuscripts for AVMA Journals

We occasionally receive requests for information on preparing manuscripts for publication in the AVMA *Journals*. To answer just such questions, an "AVMA Style Guide" was published in 1955 and a few are still available. However, recent policy changes, such as those mentioned below, make revision necessary before the "Guide" is reprinted.

For general remarks on preparing manuscripts, writers are referred to an editorial in the JOURNAL (Oct. 15, 1956: 389). In another editorial (April 1, 1957: 315) terminology was discussed. More discussions on terminology will follow at intervals. Some comments on these editorials have been published under *Correspondence*.

### NAMES OF DRUGS

As a result of the increasing tempo in the introduction of new drugs and new combinations of drugs, it has become difficult to determine which trade names are registered. Therefore, the (R) has been discontinued and the first letter of the trade name is capitalized.

Also, in an attempt to establish the use of generic or official names of drugs, the trademarked name will be used only the first time it appears in the manuscript, and again in the summary. It will appear in parentheses, following its generic or official name or its chemical designation, with a footnote giving information on the drug and the manufacturer.

Only the active ingredients of combined drugs should be mentioned in the text. However, if this is not possible or practical, the trademarked name of the combined drug will be used, as indicated above; its formula will be given in the footnote; and it will be referred to in the text as "the product," "the mixture," etc.

We realize that this policy does not present the easiest method of dealing with drug names but it should be the most informative. This method poses a particular problem when drugs are compared in tables. How closely it can be followed, only time will tell.

### REFERENCES AND AUTHORS

In both *Journals*, the reference list should be arranged alphabetically, according to the surname of the first author, and referred to in the text by a number.

In the JOURNAL of the AVMA, but not in the *American Journal of Veterinary Research*, the names of authors should be mentioned in the text only when they are directly quoted. The names are deleted for easier reading and to save space.

### JOURNAL ILLUSTRATIONS

No single feature of our *Journals* has been criticized as frequently or perhaps as justly as have photomicrographic illustrations. When details are not reproduced clearly in any illustration, it merely wastes space and frustrates the readers.

Suggested means of improving illustrations include: (1) printing the *Journals* on better paper (this would increase the costs of paper and mailing); (2) printing the illustrations on enamel paper inserts (this would increase costs and often "dislocate" the illustration); (3) insisting on better originals (authors should remember that some detail will be lost in reproduction).

When large pictures are submitted, the authors should indicate whether and how much they may be reduced, or exactly where they may be cropped to preserve the important details and to fit the columns (2⅝ in.; 4 in.; or 5½ in.). Sufficient background should be included to insure identification.

Furthermore, authors should remember that readers are usually less familiar than they with the objects illustrated. Therefore, arrows or small letters should be placed on illustrations to designate the details that are not easily recognized.

### OTHER SPECIFICATIONS

*Duplicate Copies.*—All manuscripts or articles submitted to either *Journal* should be in duplicate—the original and a clear carbon copy. They should be double spaced with ample margins (1 in. at the sides and 2 in. at top and bottom). This will allow reviewers to enter minor comments on one copy while the other is kept "clean" for the printer.

When a paper is returned to the author for revision, the original copy of all pages he retypes should be returned to us with the revised copy.

*Galleys.*—Galleys are sent to all authors to be checked for errors. Authors occasionally are indignant when errors which they had failed to correct appear in print.

ABSTRACTS

## Cultivation of Sheep Pox Virus

The Egyptian Cairo S strain of sheep pox virus was successfully cultivated on the chorioallantoic membrane of the developing chicken embryo for 21 passages, using the alternating method between sheep and chicken embryos. Until now, only three direct passages of the virus in the membrane had been attained. The amount of virus obtained from the membrane was, in some passages, equal to that given by sheep itself, as the titer reached $10^{-5}$ and $10^{-6}$. Now the embryo can be used as a cheap host for large scale production of seed virus for vaccine preparation. The cultured virus still caused generalized infection in sheep and did not cause any reaction in rabbits. The cultured virus was found to cross immunize fully with both the original Egyptian virus and the Romanian strain.

In experimental studies of the cultivation of the sheep pox virus in the egg, it was found that two, three, or four days' incubation after inoculation was the best time for obtaining the maximum amount of virus from the membrane, that the optimum temperature was between 36 and 37 C. No virus grew at 39 C. The highest titer of virus was found in the chorioallantoic membrane (c.a.m.) and the least was found in the embryo and fluids. The virus produced characteristic lesions in the c.a.m. in the form of thickened, yellow, caseated patches, of different sizes, at the area of inoculation. Microscopically, the ectodermal layer showed proliferation, degeneration, and necrosis with considerable hypertrophy of the mesoderm. Several attempts were made to cultivate the Romanian strain of sheep pox virus on the c.a.m. using different methods with unsuccessful results. —[*M. S. Sabban: The Cultivation of Sheep Pox Virus on the Chorioallantoic Membrane of the Developing Chicken Embryo. Am. J. Vet. Res., 18, (July, 1957): 618-624.*]

## Elementary Bodies of Sheep Pox

Paschen's method was found to be the best for staining elementary bodies of sheep pox virus in purified suspensions as well as smears of skin lesions. In electron micrographs, the virus was found to resemble other pox viruses but was smaller and more elongated. It appeared to have subpolar densities like the virus of contagious ecthyma. No central dense spot could be demonstrated. Average dimensions were: length 194 $\pm$16.08 $\mu$, breadth 115 $\pm$10.73 $\mu$, axial ratio (length/breadth) 1.76 $\pm$0.176.—[M. Abdussalam: *Elementary Bodies of Sheep Pox. Am. J. Vet. Res., 18, (July, 1957): 614-617.*]

## Chlorinated Naphthalene Intoxication in Sheep

Experimental highly chlorinated naphathalene intoxication was produced in three groups of 5 sheep each. Sheep in one group were given 0.5 mg. of the chemical per pound of body weight daily, an average total of 53.0 mg./lb. in 106 days, resulting in death of the sheep. Those in the second group were given 5.0 mg./lb. of body weight daily, an average total of 137.0 mg./lb. in 27 days, causing death of the sheep. Those in the third group were given 12.5 mg./lb. of body weight daily, an average total of 256.0 mg./lb. in 20 days, also resulting in death of the sheep. This indicates that sheep have a much greater tolerance for the chemical than do cattle. These sheep did not show cutaneous hyperkeratosis or an excessive drop in plasma vitamin A level. Affected sheep showed nasal discharge, weakness, loss of weight, inappetence, and ascites. The pathological findings were those associated with cardiovascular injury, necrosis and cirrhosis of the liver, and damage to the nephrons.—[*W. E. Brock, E. W. Jones, Robert MacVicar, and L. S. Pope: Chlorinated Napthalene Intoxication in Sheep. Am. J. Vet. Res., 18, (July, 1957): 625-630.*]

## Concentrations of Phenothiazine in Blood Serum, Bile, and Urine

Maximum drug levels based on a 100-lb. live body weight (137.8 $\mu$g. of phenothiazine and 342.0 $\mu$g. of phenothiazone) occurred at 24 hours post-treatment. Maximum drug levels in bile (72.9 mg. of phenothiazine and 64.8 mg. of phenothiazone) were detected at 16 hours. Maximum drug levels in the urine (2.40 mg. of phenothiazine/ml. and 10.80 mg. of phenothiazone/ml.) were detected at 20 hours. No traces of drug were detected in blood serum, bile, or urine at 120 hours post-treatment. —[*T. Ellison and A. C. Todd: II. Chronological Levels of Drug in Blood Serum, Bile, and Urine. Am. J. Vet. Res., 18, (July, 1957): 522-525.*]

## Chronological Levels of Phenothiazine in Organs

The first appearance of drug in the liver was phenothiazone at two hours post-treatment. Phenothiazine was detected at three hours. Maximum drug levels were at 16 hours for phenothiazone and 24 hours for phenothiazine. The chronology of drug appearance in the kidneys was the reverse of the situation existing in the liver. Maximum drug levels in the spleen occurred at five hours for phenothiazine and phenothiazone. In the mesenteric lymph nodes, maximum drug levels occurred at 12 hours for phenothiazone and 14 hours for phenothiazine. No drug was detected in liver, kidneys, spleen, or mesenteric lymph nodes at 120 hours post-treatment.—[*T. Ellison and A. C. Todd: III. Chronological Levels of Drug in Liver, Kidney, Spleen, and Mesenteric Lymph Nodes. Am. J. Vet. Res., 18, (July, 1957): 526-527.*]

## Recovery of Phenothiazine, N. F., in Dairy Calves

Eighteen grade dairy calves were each dosed orally with 100 Gm. of phenothiazine, N. F. Total

concentrations of phenothiazine and phenothiazone were given for the following post-treatment periods: 1, 2, 3, 4, 5, 6, 8, 10, 12, 14, 16, 18, 20, 22, 24, 40, 80, and 120 hours. No drug was detected at 120 hours post-treatment.—[*T. Ellison and A. C. Todd: IV. Chronological Total Drug Recovery. Am. J. Vet. Res., 18, (July, 1957): 527-529.*]

## Phenothiazine Treatment of Weaned Calves

Four experiments involved 81, 16, 11, and 8 calves, respectively. Calves of experiments 1 and 2 were given therapeutic doses of phenothiazine at the beginning; those of 3 and 4 were not. Calves of the medicated group of experiment 1 received 1 Gm. of phenothiazine daily per calf and gained the same amount of weight as the controls. Calves of the medicated groups of experiment 2, 3, and 4 received 2 Gm. of phenothiazine daily per calf and gained 60, 25, and 35 lb. more, respectively, than the controls. The worm eggs per gram of feces of the control groups averaged 26, 44, 125, and 56 per cent greater than those of the medicated groups.—[*G. E. Cauthen: Low Level Phenothiazine Administration to Weaned Beef Calves. Am. J. Vet. Res., 18, (July, 1957): 612-613.*]

## Weight Gain in Phenothiazine-Treated Calves

In ten separate experiments, over a seven-year period, unweaned and weaned Hereford, Brahman, and mixed Hereford-Brahman calves were given single doses (54 to 96 Gm.) of green, NF grade phenothiazine at various times from 68 days before, to 90 days after, weaning. In the one experiment involving heavy subclinical parasitism in unweaned calves, the 59 calves gained an average of 22 lb. during the 97-day test period. The 14 controls lost 11 lb. The other nine experiments, involving calves with moderate subclinical parasitism, could be divided into four groups. In one group of unweaned calves, 149 treated calves gained an average of 5 lb. more than the 154 controls; 499 treated calves of the remaining 3 groups gained 3 lb. less than the 410 controls. Toxic effects of phenothiazine for calves or development of immunity by the parasites were suggested as possible factors responsible for the low weight gain in treated calves.—[*G. E. Cauthen: Weight Gain in Phenothiazine-Treated Calves. Am. J. Vet. Res., 18, (July, 1957): 608-611.*]

## Isolation of Vibrio Fetus from Bulls

A procedure for detecting bulls which carry *Vibrio fetus* by culturing semen and perputial washings on blood agar, incubating the plates in the optimum gaseous environment for *V. fetus*, and isolating and identifying the organism is described. The incidence of carrier bulls in 4 A. I. centers was 27, 34, 40, and 57 per cent (av. = 41% of 149 bulls.) The percentage of carrier bulls found positive by 1, 2, and 3 tests was 39, 67, and 95 per cent. In repeated tests on 57 carrier bulls, 54 per cent of 263 semen samples yielded *V. fetus;* the rate of isolating *V. fetus* from individual bulls varied from 1 of 6 to 6 of 6 tests.—[*W. N. Plastridge, E. C. Walker, L. F. Williams, E. F. Stula, and E. M. Kiggins: Isolation of Vibrio Fetus from Bulls. Am. J. Vet. Res., 18, (July, 1957): 575-578.*]

## Pathology of Virus Diarrhea in Indiana Cattle

Examinations of field infected animals which had died of virus diarrhea, and field and experimental animals slaughtered in the acute, chronic, or recovery phases revealed changes in the mucosa of the alimentary canal and the lymphatic tissue. Lesions of the alimentary canal were primarily of an erosive and ulcerative nature and were associated with edema, congestion, and hemorrhage, particularly in those areas where only a single cell layer separates the lumen of the tract from the underlying vascular stroma. Edema and exhaustion of the germinal centers were the principal changes observed in lymphatic tissue.—[*R. G. Carlson, W. R. Pritchard, and L. P. Doyle: The Pathology of Virus Diarrhea of Cattle in Indiana. Am. J. Vet. Res., 18, (July, 1957): 560-568.*]

## Electronic Stethoscope for Sterile Surgery

The design and construction of a relatively inexpensive electronic stethoscope for use in sterile small animal surgery is described. The instrument employs a compensated amplifier providing three bandwidths, a cathode follower output for auscultation, and a triggering circuit which controls a neon bulb for visual indication. The circuitry, application, and advantages during surgery, where sterile draping complicates direct monitoring, are discussed.—[*J. A. Davis, B. D. Fremming, and H. M. Borella: An Electronic Stethoscope for Sterile Small Animal Surgery. Am. J. Vet. Res., 18, (July, 1957): 634-637.*]

# BOOKS AND REPORTS

## The Future of Arid Lands

In the spring of 1955, the International Arid Lands Meeting, comprising representatives from 17 countries, met in New Mexico to discuss the future of arid and semiarid lands. The papers, which are published in this volume, covered the subjects of water supply, better use of current water and grazing resources, and plant and animal adaptation. The meeting pointed out the need of making scientific information available to farmers, herders, and landowners of semiarid areas; the need for further research in reclamation of lands made semiarid through centuries of misuse by man; and suggested methods whereby these lands might be restored while maintaining the human and animal populations dependent on them.—[*The Future of Arid Lands. Edited by Gilbert F. White. 453 pages. Illustrated. American Association for the Advancement of Science, Washington, D. C. 1956. Price not given.*]

# THE NEWS

## Conference on Animal Disease and Human Health Held in New York

A conference on Animal Disease and Human Health was held at the Grand Ballroom of the Hotel Delmonico in New York City, Sept. 11-13, 1957. It was sponsored by the New York Academy of Sciences, Section on Biology and the United States Public Health Service, Communicable Disease Center, Atlanta, Ga. The three-day session was directed by Dr. James Lieberman, Atlanta.

Some of the speakers and their corresponding subjects are: J. D. Martin, Louisiana State Health Department, New Orleans—Animal Diseases—Their Relationship to the Health of Man; Lt. Col. Jack H. Hempy, Office of the Surgeon General, U.S.A.F., Washington—Animal Disease and Biological Warfare Defense; Roy W. Chamberlain, Communicable Disease Center, Montgomery—Vector Relationships in the Arthropod-Borne Encephalitides; B. Babudieri, Instituto Superióre di Sanità, Rome, Italy—Animal Reservoirs; Albert A. Benedict, University of Kansas, Lawrence—Newer Methods for Detection of Avian Ornithosis; M. L. Kaeberle, University of Illinois, Champaign—Newer Tools for the Prevention of Rabies in Domestic Animals; John P. Fox, Tulane University, New Orleans—Prophylaxis Against Rabies in Humans—1957; Robert J. Anderson, Communicable Disease Center, Atlanta—Public Health Importance of Animal Tuberculosis; and James A. Prier, Merck Sharp & Dohme, West Point, Pa.—Cat Scratch Fever.

## National Cattle Grub Committee Created at Kansas City

Experts representing every segment of the livestock industry met at Kansas City, Mo., in August, 1957, to tackle the many problems incident to a national cattle grub control and eradication program. This workshop sponsored by Livestock Conservation, Inc., resulted in the creation of a national cattle grub committee.

This committee will be divided into four subcommittees: Research and Evaluation; Economics of Grub Control; Education and Information; Program and Procedures.

The keynote speaker at the meeting declared that "cattle grub . . . costs the cattle industry well over $100,000,000 annually, . . . and is present in every state." Many men in the field consider this disease as an economic loss about which nothing can be done.

Research workers have been working toward the elimination of this parasitic pest, but it was not until the end of World War II that some momentum in the project increased. The systemic chemicals which have been developed are not presently available for general use, due to the "residue" in both the tissue and milk of treated animals.

Dr. J. W. Cunkelman, chief veterinarian, Swift & Co., was named as permanent chairman of the general committee, and R. Harvey Dastrup, general manager, Livestock Conservation Inc., as permanent secretary.

## Symposium on Zoonoses in the Tropics

A symposium on Zoonoses in the Tropics will be held in connection with the joint meeting of the American Society of Parasitologists and the American Society of Tropical Medicine and Hygiene in Philadelphia, Oct. 30 to Nov. 2, 1957.

Interested veterinarians are invited to attend the symposium which is scheduled for Thursday, Oct. 31, 1957, at 2:00 p.m. in the Benjamin Franklin Hotel.

The program will be moderated by Dr. Willard H. Wright of the National Institutes of Health. The speakers and their subjects are: Drs. James H. Steele, Communicable Disease Center, Atlanta, —Some Zoonoses Which Constitute Important Health Problems in the Tropics; Aurelio Malaga-Alba, Pan-American Scientific Bureau, Mexico,—Tropical Bat Rabies as a Public Health and Veterinary Problem; Morris Pollard, University of Texas Medical School, Galveston,—Tropical Arthropod-borne Viruses—Their Importance in Veterinary and Human Medicine; and D. G. Davey, Imperial Chemical Industries, Ltd., England,—Human and Animal Trypanosomiasis in Africa.

## Dr. Elmer Hess Appointed Chairman of Health Resources Advisory Committee, Office of Defense Mobilization

Gordon Gray, director of the Office of Defense Mobilization, announced Aug. 1, 1957, the appointment of Elmer Hess, M.D., Erie, Pa., as chairman of the O.D.M. Health Resources Advisory Committee. He succeeds Dr. Howard A. Rusk, New York City.

Dr. Hess is a past-president of the American Medical Association, and of the American Urological Association. He served in France with the Army Medical Corps during World War I and has performed several missions for the government overseas.

## Drs. L. C. Harold and F. J. Kingma Join the Professional Staff of the Food and Drug Administration

Two veterinarians have joined the Food and Drug Administration of the Department of Health, Education, and Welfare. Dr. LaVerne C. Harold reported for duty on June 25, 1957, and Dr. Fred J. Kingma joined the professional staff of the Food and Drug Administration on July 29, 1957.

Dr. Kingma was granted his D.V.M. degree in 1938 and his master of science degree in 1939 from Ohio State University. From 1946 to 1955, Dr. Kingma was secretary of the Ohio State Veterinary Medical Association. He has served as Ohio's delegate to the AVMA House of Representatives and to the AVMA Executive Board from District X. In his new post, Dr. Kingma will be concerned with the problems encountered in the enforcement of the federal Food, Drug, and Cosmetic Act.

Dr. Harold received his B.S. degree in Agriculture, and his D.V.M. degree from Kansas State University in 1945. Subsequently, he obtained a bachelor of laws degree (LL.B.) from the University of Kansas City. He will specialize in the use of various antibiotic drugs in the animal therapy field.

## AMONG THE STATES AND PROVINCES

### Alabama

**Dr. R. S. Sugg Honored at Alabama Polytechnic Institute's Annual Conference.**—At the fiftieth annual veterinary conference held July 21-24, 1957, at Alabama Polytechnic Institute in Auburn, a painting of Dean Sugg was presented to the school by Dr. W. S. Bailey for the veterinary faculty as a token of appreciation for Dr. Sugg's work and achievements.

Dr. Sugg is a member of many honorary and professional organizations from which he has received numerous awards and honors. Among these are certificates in recognition of services as president of the Alabama V.M.A. and the Southern V.M.A. as well as the Twelfth International Veterinary Congress Prize for his contribution to the development of the regional program for veterinary education. He is a member of Alpha Psi Fraternity, Epsilon Sigma Phi, Phi Kappa Phi, O. D. K., Masons, Kiwani, Phi Zeta, the AVMA, United States Livestock Association, Alabama Veterinary Medical Association, and the Association of Military Surgeons.

**Dr. R. S. Sugg (second from right) accepting his portrait for the school from Dr. W. S. Bailey (right) at Alabama Polytechnic Institute's Fiftieth Annual Conference.**
**Drs. J. E. Greene (left) and C. H. Clark (third from right) also participated in the ceremonies.**

Dr. Sugg is currently serving a second term as member of the Executive Board of the AVMA from District IV.

### California

**A Course on Veterinary Radiology, Pathology, and Endocrinology Offered at the University of California Medical Center.**—University of California Extension and the U.C.L.A. School of Medicine will cooperate with the Southern California V.M.A. to offer a course in "Selected Topics in Veterinary Radiology, Pathology and Endocrinology" at the university's medical center this fall from October 3 to Nov. 21, 1957, on Thursdays at 7:30 to 9:30 p.m.

The course is open only to graduates of approved schools of veterinary medicine with the consent of the course chairman, Bennett J. Cohen, D.V.M., Ph.D. Moses A. Greenfield, Ph.D., will deliver the initial October 3 lecture on "X-ray Theory and Safety".

Requests for applications or information should be made to Thomas H. Sternberg, M.D., assistant dean, Postgraduate Medical Education, University of California Medical Center, Los Angeles 24.

### Illinois

**University of Illinois Animal Hospital to Hold Lecture Series.**—The Animal Hospital of the University of Illinois Professional Colleges (dentistry, medicine, pharmacy) will sponsor a lecture series to be presented during the fall quarter on each Friday from October 11 to Dec. 13, 1957, at 12 noon to 1:00 p.m., in Room 608, 1853 West Polk St., Chicago.

An invitation is extended to all members of the AVMA to attend these lectures. Following is a list of the speakers and subjects:

**October 11**—Respiratory Virus Diseases of Fowl and the Status of Vaccines Against These Diseases, Dean C. A. Brandly, D.V.M., M.S., College of Veterinary Medicine, University of Illinois, Urbana.

**October 18**—The Clinical Symptoms and Treatment of Diseases of Zoo Animals, Dr. L. E. Fisher, D.V.M., Berwyn Animal Hospital, Berwyn; and attending veterinarian, Lincoln Park Zoo, Chicago.

**October 25**—Residual Toxicity of Newer Insecticides in Animal Tissue, Dr. R. P. Link, D.V.M., Ph.D., Department of Physiology

and Pharmacology, College of Veterinary Medicine, Urbana.

**November 1**—Endemic Diseases of Farm Animals in the Chicago Area which are Contagious to Humans, Dr. R. Galvin, D.V.M., Beecher.

**November 8**—Diseases of Dogs, Comparable to Human Diseases, Which Can Be Diagnosed by Radiography, Dr. D. Maksic, D.V.M., Department of Small Animal Medicine, College of Veterinary Medicine, Urbana.

**November 15**—Psittacosis in Laboratory Fowl and Its Diagnosis, Dr. L. E. Hanson, Department of Pathology, College of Veterinary Medicine, University of Illinois, Urbana.

**November 22.** (12 noon to 2 p.m. a lecture and movie)—A Complete Hip Prosthesis Developed in Dogs and Adapted for Human Use, Col. H. Gorham, D.V.M., U.S.A.F., Randolph Field, Texas.

**November 29**—The Incidence and Control of Rabies in Animals in Cook County, Illinois, for the Past Five Years (a movie of rabies in the human will also be shown), Dr. E. C. Khuen, D.V.M., Cook County rabies Inspector, Chicago.

**December 13**—Veterinary Clinical Research, Dr. G. Burch, D.V.M., director of Veterinary Research, Pitman-Moore Co., Indianapolis, Ind.

s/WILLIAM C. DOLOWY.

## Maryland

**State Association.**—At the summer meeting of the Maryland State Veterinary Medical Association at Ocean City on June 28, 1957, the following officers were elected for the coming year: Drs. Dale A. Wiley, Jarrettsville, president; Walter J. Hastings, Jr., Cambridge, first vice-president; Donald R. Lynch, Frederick, second vice-president; Harry L. Schultz, Jr., Baltimore, secretary-treasurer.

The Association's board of directors, elected at the same meeting, are: Drs. Robert M. Lynn, Riverdale; Robert C. Flaherty, Easton; Charles G. Ziegler, Cantonsville; Edward I. Neserke, Baltimore; and Arthur L. Brueckner, Hyattsville.

s/HARRY L. SCHULTZ, JR., *Secretary.*

## Montana

**Women's Auxiliary.**—The annual meeting of the Montana V.M.A. was held June 28, 1957, in the Assembly Room of the Northern Hotel, Billings.

Mrs. Glenn Halver, Glendive, presided at the meeting. A report on the national meeting in San Antonio was presented by Mrs. Hadleigh Marsh, Bozeman. It was voted to make contributions to the National Loan Fund, the Research Fund, as well as to give a $100 scholarship to a worthy Montana student in a recognized veterinary college.

The following officers were elected for the ensuing year: Mrs. I. W. Vinsel, Dillon, president; Mrs. D. K. Shaffner, Dillon, president-elect; Mrs. R. J. Miser, Conrad, first vice-president; Mrs. J. H. Newhall, Bozeman, second vice-president; Mrs. S. D. Gates, Lewistown, third vice-president; Mrs. O. W. Bakke, Columbus, secretary; and Mrs. D. G. Colmey, Livingston, treasurer.

## New Hampshire

**State Association.**—The summer meeting of the New Hampshire Veterinary Medical Association was held at the Wentworth-by-the Sea on July 24, 1957.

The program was arranged by Dr. Lionel Trudel of Portsmouth, and consisted of the following speakers and subjects: Drs. Sydney Stiles, Falmouth Foreside, Portland, Maine—Highlights of Large Animal Practice; Patricia O'Connor, Staten Island Zoo, New York—Care of Wild Animal Pets; George Ott, Fromme Laboratories, Grafton, Wis.—New Concepts of Canine Distemper Vaccination.

s/FRED E. ALLEN, *Secretary.*

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**Women's Auxiliary.**—The Women's Auxiliary to the New Hampshire V.M.A. met in the Tally-Ho room of the Wenworth-by-the Sea in conjunction with the meeting of the State Association on July 24, 1957.

Mr. Herman Page, Portsmouth, a certified Gemmologist and a registered jeweler of the American Gem Society was the guest speaker. Mrs. Alfred E. Coombs, Skowhegan, Maine, then president of the AVMA Women's Auxiliary, was the guest of honor.

## North Dakota

**State Association.**—The annual meeting of the North Dakota Medical Association was held June 16-18, 1957, at the Clarence Parker Hotel in Minot.

The Association's guest speakers included: Dr. W. Beckenhauer, Norden Laboratories; Dr. W. W. Armistead, College Station, Texas; C. K. Mingle, Washington, D.C.; Dr. H. Ross, Brandon, Manitoba; Dr. P. E. Madsen, Sheridan, Wyo.; and C. P. Lura, president of the Minot State Teacher's College.

At the meeting, it was decided to honor with life memberships all veterinarians who had been continuous members of the Association for 40 years or more. The following veterinarians received this honor at the banquet held during the course of the three-day meeting: Drs. T. O. Brandenburg, Bismarck; J. C. Jirikowic, Bottineau; J. V. Miles, Ellendale; J. W. Robinson, Garrison; R. E. Shigley, Minot; and J. H. Winslow, La Moure.

The officers elected for the coming year are: F. L. Whitcomb, Bismarck, president; D. K. Christian, Moorhead, Minn., vice-president; Dean E. Flagg, Bismarck, secretary-treasurer, and resident secretary.

s/DEAN E. FLAGG, *Secretary-Treasurer.*

**Women's Auxiliary.**—The Women's Auxiliary to the North Dakota V.M.A. elected the following officers for the ensuing year: Mrs. S. S. Bjornson, West Fargo, president; Mrs. F. M. Bolin, Fargo, vice-president; Mrs. L. G. Best, Wahpeton, secretary; and Mrs. R. C. Reed, Hillsboro, treasurer.

## U. S. GOVERNMENT

**Veterinary Personnel Changes.**—The following changes in the force of veterinarians in the U.S.D.A. are reported as of July 31, and Aug. 1, 5, and 6, 1957.

TRANSFERS

Charles C. Bickley, from Helena, Mont., to Reno, Nev.
Robert N. Birdwhistell, from Fort Dodge, Iowa, to St. Joseph, Mo.
James E. Boyd, from National Stock Yards, Ill., to Kansas City, Kan.
William N. Dudley, from Pierre, S. Dak., to St. Paul, Minn.
Wayne M. Frerichs, from Pierre, S. Dak., to Omaha, Neb.
Ross W. Gerding, from Alexandria Bay, N.Y., to Columbia, S. Car.
Oswald H. Graham, from St. Louis, Mo., to Dallas, Texas.
Fleetwood Hines, from Jackson Miss., to Raleigh, N. Car.
Edward M. Joneschild, from Helena, Mont., to Albuquerque, N.M.
Leland B. Kendall, from Springfield, Mo., to Kansas City, Kan.
Robert E. Kind, from Jefferson City, Mo., to Kansas City, Kan.
Melvin F. Kirchhoff, from Pierre, S. Dak., to St. Paul, Minn.
Norvan L. Meyer, from Indianapolis, Ind., to Springfield, Ill.
David E. McKnight, from Sioux City, Iowa to Kansas City, Kan.
Gerald J. Miller, from Springfield, Mo., to Milwaukee, Wis.
Paul K. L. Schmidt, from National Stock Yards, Ill., to St. Louis, Mo.
Louis M. Singleton, from Bismarck, N. Dak., to St. Louis, Mo.
Paul O. Thomas, from Springfield, Mo., to Milwaukee, Wis.
Donald L. Welch, from Jefferson City, Mo., to St. Paul, Minn.
Edward J. Wilson, from Columbus, Ohio, to Springfield, Ill.
James H. Wommack, from Olympia, Wash. to Cheyenne, Wyo.

RETIREMENTS

Howard A. Allen, Los Angeles, Calif.
Thomas A. Head, Milwaukee, Wis.
David C. Wilke, Portland, Oregon.

DEATHS

John T. E. Dinwoodie, Billings, Mont.

## DEATHS

**Star indicates member of AVMA**

**George H. Davidson** (ONT '98), Rugby, N. Dak., died on Nov. 7, 1956. He was born at LeSueuer, Minn., and he attended the Chicago Veterinary College and Toronto Veterinary College.

Dr. Davidson's wife preceded him in death in 1946.

**Harry Rushton** (ONT '41), 40, died in Berlin, Germany, on Aug. 2, 1957.

Captain Rushton was born in England, and after graduation, he conducted a practice in Flint, Mich. He was on active duty as an officer of the Army Veterinary Corps since 1951. At the time of his death, he was assigned to duty in Europe.

He is survived by his widow and two children.

**Levi P. Sterling** (ISC '19), 65, Zearing, Iowa, died at Mercy Hospital in Marshalltown sometime in the summer of 1957.

He had been a government inspector at Zearing for the last four years. Dr. Sterling is survived by his widow and a daughter.

★**B. Lamar Watson** (API '48), 34, Marianna, Fla., died early in 1957 of cancer. Dr. Watson had been a member of the Florida Veterinary Medical Association. He is survived by his widow, Mary.

• • •

**Other Deaths Reported.**—The following deaths have been reported. The usual information for an obituary was not supplied.

Derwin W. Ashcraft (OSU '49), Washington, D.C., died late in 1955.

Charles W. Barrett (NYS '05), Pasadena, Calif.

Georges Beauchemin (MON '17), Quebec City, Que.

Howard S. Boggs (OSU '04), Kingston, Ohio.

Hurst M. Cameron (ISC '11), Alden, Minn.

Charles A. Clark (MCK '10), Oswego, Ill.

John S. Evans (ONT '87), Clinton, Ont.

Charles B. Frost (CVC '13), Georgetown, Ohio.

G. B. Jerdan (API '19), Gordo, Ala.

Augustus S. Martin (KCV '17), St. Joseph, Mo.

Arthur McConnell (OSU '07), Pittston, Pa.

Thomas W. McMahon (ONT '14), Tillsonburg, Ont.

George W. Meyer (AVC '91), Gloversville, New York.

Stanley L. Pilgrim (CVC '11), Milwaukee, Wis.

I. Peters (TH '11), Summerfield, Fla.

John W. Steinbrook (SF '16), Modesto, Calif.

Winters D. Stockstill (CIN '09), Sidney, Ohio.

Catala Juan Varas (OSU '10), Arecibo, Puerto Rico.

Milton B. West (ONT '05), Ryley, Alberta, Can.

L. M. Wright (CVC '09), Waukesha, Wis.

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*Treasurer*—H. E. Kingman, Jr., *ibid.*

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*At Large*—Martin T. Hines, 1729 Brooks Avenue, Raleigh, N. Car.

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*District II*—S. F. Scheidy, 943 Turner Ave., Drexel Hill, Pa. (1958).

*District III*—L. M. Hutchings, *ibid.* (1958).

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*District VI*—Joseph M. Arburua, 3020 26th Ave., San Francisco, Calif. (1961).

*District VII*—E. C. Stone, College of Veterinary Medicine, State College of Washington, Pullman, Wash. (1960).

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*District IX*—Myron G. Fincher, New York State Veterinary College, Ithaca, N. Y. (1962).

*District X*—Wade O. Brinker, College of Veterinary Medicine, Michigan State University, East Lansing, Mich. (1959).

*Ex officio*—W. W. Armistead, College of Veterinary Medicine, Michigan State University, East Lansing, Mich. (1959).

*Ex officio*—R. E. Rebrassier, College of Veterinary Medicine, Ohio State University, Columbus, Ohio. (1960).

*Ex officio*—Wayne O. Kester, 4846 Bethesda Ave., Bethesda, Md. (1958).

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L. M. Hutchings, *Chairman;* W. W. Armistead; R. E. Rebrassier.

### Advisory Committee of the House of Representatives

*Chairman*—W. T. Oglesby, Department of Veterinary Science, University of Louisiana, Baton Rouge 3, La.

*Representing Large Animal Practice*—C. A. Bjork, 3644 N. E. Flanders, Portland, Ore. (1960).

*Representing Large Animal Practice*—A. E. Coombs, P.O. Box 174, Skowhegan, Maine. (1959).

*Representing Mixed Practice*—Niel Pieper, Randolph Rd., Middletown, Conn. (1959).

*Representing Small Animal Practice*—J. O. Knowles, 2102 N.W. 25th Ave., Miami 42, Fla. (1958).

*Representing Teaching and Research*—W. T. Oglesby, *ibid.* (1958).

*Representing Federal or State Government Regulatory Services*—A. P. Schneider, 2025 N. 23rd St., Boise, Idaho (1959).

*Representing the Armed Forces and the U. S. Public Health Service*—Elmer W. Young, Office of the Surgeon General, Main Navy Bldg., 18th and Constitution Ave., N.W., Washington 25, D.C. (1960).

### Headquarters

600 South Michigan Ave., Chicago 5, Ill.

### Executive Staff

*Executive Secretary*—J. G. Hardenbergh, V.M.D.

*Assistant Executive Secretary and Director of Professional Relations*—H. E. Kingman, Jr., D.V.M.

*Editor-in-Chief*—W. A. Aitken, D.V.M.

*Director of Membership Services and Business Manager*—R. G. Rongren.

*Director of Public Information*—B. M. Forster.

### Secretarial Staff

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*Librarian*—Gertrude Schwerin.

*Receptionist*—Josephine Freund.

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*Assistant Editor in Charge of Production*—Helen S. Bayless.

*Advertising Manager*—Helen S. Bayless.

*Assistant to the Editor*—Eva G. Bailey.

*Assistant Advertising and Production Manager*—Dinah Polen.

*Editorial Assistants*—Marilyn Ott and Mary Parks.

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J. A. McCallam, V.M.D. (Brig. Gen., Ret.), Rm. 109, 1507 M St., N.W., Washington 5, D.C. Telephone: NOrth 7-0881.

### Associate Editors

*Diseases of Horses*—P. J. Meginnis, R. R. 1, Roselle, Ill.

*Diseases of Dairy Cattle*—S. J. Roberts, New York State Veterinary College, Ithaca, N.Y.

*Diseases of Beef Cattle*—H. W. Johnson, 5000 S. Santa Fe St., Littleton, Colo.

*Diseases of Swine*—J. D. Ray, White Hall, Ill.

*Diseases of Sheep and Goats*—Hadleigh Marsh, 916 S. Third St., Bozeman, Mont.

*Diseases of Poultry*—Henry Van Roekel, Department of Veterinary Science, University of Massachusetts, Amherst.

*Diseases of Small Animals*—K. W. Smith, 1002 34th St., Sioux City, Iowa.

*Diseases of Wildlife and Captive Animals*—L. E. Fisher, 2823 S. Harlem Ave., Berwyn, Ill.

*Diseases of Laboratory Animals*—W. T. S. Thorp, College of Veterinary Medicine, University of Minnesota, St. Paul 1, Minn.

*U.S.D.A.*—C. D. Van Houweling, Assistant Administrator, ARS, U.S.D.A., Washington 25, D.C.

*Public Health Services*—Raymond Fagan, 11 Locust St., Goshen Heights, West Chester, Pa.

*Military Services*—E. W. Young, Veterinary Division, Office of the Surgeon General, Department of the Army, Main Navy Bldg., Washington, 26, D.C.

### Section Officers

(These officers also constitute the Committee on Program with the executive secretary or assistant executive secretary of the AVMA as chairman, ex officio.)

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*Poultry.*—P. P. Levine, *Chairman,* New York State Veterinary College, Ithaca, N.Y.; M. S. Cover, *Secretary,* Agricultural Experiment Station, University of Delaware, Newark, Del.

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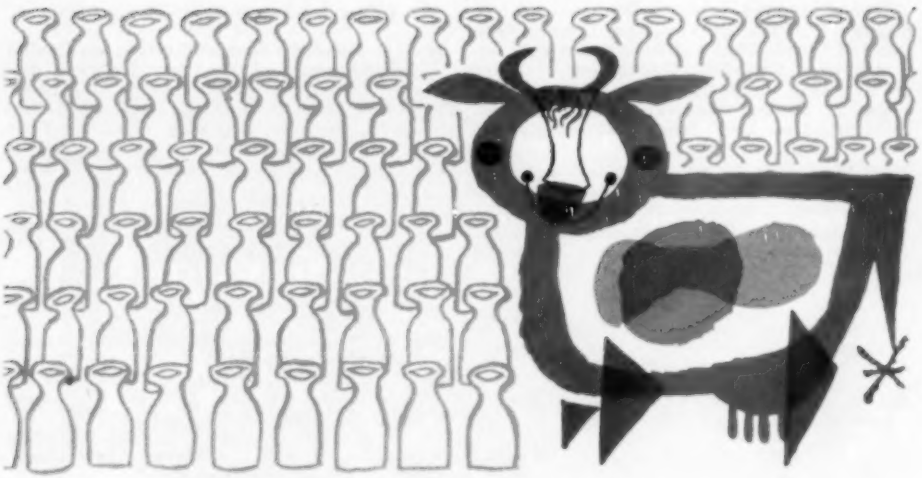
R. E. Rebrassier, as President-Elect of the AVMA, Veterinary Laboratory, Ohio State University, Columbus, Ohio.

L. M. Hutchings, as Executive Board Chairman of



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Elmer W. Young, as Chief of the Veterinary Division, Office of the Surgeon General, Department of the Army, Washington 25, D.C.

M. R. Clarkson, as Deputy Administrator of Agricultural Research Service, U.S.D.A., Washington 25, D.C.

### Biological Products

Carl J. Norden, Jr., *Chairman,* Norden Laboratories, Lincoln, Neb. (1959).

A. L. Brueckner, 4111 Colesville Rd., Hyattsville, Md. (1958).

Ben R. Burmester, U.S. Reg. Poultry Research Laboratory, P.O. Box 270, East Lansing, Mich. (1958).

Irving G. Cashell, 2916 M St., N.W., Washington 7, D.C. (1959).

John M. Hejl, 2908 Old Dominion Building, Alexandria, Va. (1958).

William J. Zontine, 44848 N. Yucca Ave., Lancaster, Calif. (1958).

### Board of Trustees—Research Fund

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### Ethics

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Lloyd G. Best, P.O. Box 816, Wahpeton, N. Dak. (1958).

E. L. Taylor, Warrendale Ave., Georgetown, Ky. (1959).

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I. B. Boughton, Representing Research and Education, School of Veterinary Medicine, Texas A. & M. College, College Station, Texas (1958).

Col. William E. Jennings, Representing Military Service, Medical Division, Hq. USAREUR, APO 403, New York, N. Y. (1962).

T. Lloyd Jones,* Representing Basic Sciences, Ontario Veterinary College, Geulph, Ont. (1960).

Fred B. Ogilvie, Representing Small Animal Practice, 1110-12 Minnesota Ave., Kansas City 2, Kan. (1960).

Niel W. Pieper,* Representing General Practice, Randolph Rd., Middletown, Conn. (1958).

M. S. Shahan, Representing Government Service, Plum Island Animal Disease Laboratory U.S.D.A., Greenport, L.I., N.Y. (1961).

H. J. Stafseth, Representing Public Health, College of Veterinary Medicine, Michigan State University, East Lansing, Mich. (1963).

E. E. Slatter, Representing Large Animal Practice, 205 Prairie St., Danville, Ill. (1959).

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R. K. Anderson, College of Veterinary Medicine, University of Minnesota, St. Paul, Minn. (1959).

W. D. Nettles, Quartermaster Food and Container Institute for the Armed Forces, 1819 West Pershing Rd., Chicago 9, Ill. (1959).

M. P. Hines, Veterinary Public Health Section, State Board of Health, Raleigh, N. Car. (1958).

F. P. Jaggi, Jr., School of Veterinary Medicine, Texas A. & M. College, College Station, Texas (1958).

C. H. Pals, Meat Inspection Branch, Agricultural Research Service, U.S.D.A., Washington 25, D.C. (1958).

### History

J. F. Smithcors, *Chairman,* College of Veterinary Medicine, Michigan State University, East Lansing, Mich. (1959).

J. M. Arburua, 3020 26th Ave., San Francisco, Calif. (1958).

H. P. Hoskins, 2766 Garrison Ave., Evanston, Ill. (1958).

A. A. Kingscote, Department of Parasitology, Ontario Veterinary College, Guelph, Ont. (1958).

A. H. Quin, P.O. Box 167, Kansas City, Mo. (1959).

Hubert Schmidt, 712 Eagle Pass, Bryan, Texas.

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Lloyd C. Moss, Veterinary Hospital, Colorado State University, Fort Collins, Colo. (1960).

### Legislation

F. B. Young, *Chairman,* Box 6, Waukee, Iowa (1958).
J. A. McCallam, Room 109, 1507 M St., N.W., Washington, D.C. (1958).
N. J. Miller, P.O. Box 335, Eaton, Colo. (1958).
S. F. Scheidy, 943 Turner Ave., Drexel Hill, Pa. (1958).
Elmer W. Young, Veterinary Division, Office of the Surgeon General, Department of the Army, Washington 25, D.C. (1958).

### Nutrition

C. K. Whitehair, *Chairman,* College of Veterinary Medicine, Michigan State University, East Lansing, Mich. (1958).
William D. Pounden, 625 Sunrise View Dr., Wooster, Ohio (1958).
Robert W. Dougherty, New York State Veterinary College, Ithaca, N.Y. (1958).
Raymond T. Hander, Bridwell Hereford Ranch, Windthorst, Texas (1958).
R. E. Lubbehusen, Purina Mills, 835 S. 8th St., St. Louis 2, Mo. (1958).
R. H. Udall, College of Veterinary Medicine, Colorado State University, Fort Collins, Colo. (1958).

### Parasitology

L. E. Swanson, *Chairman,* 1325 N.W. 14th Ave., Gainesville, Fla. (1958).
L. P. E. Choquette, École de Médecine Vétérinaire, St. Hyacinthe, Que. (1958).
F. R. Koutz, College of Veterinary Medicine, Ohio State University, Columbus 10, Ohio (1959).
Wendell Krull, College of Veterinary Medicine, Oklahoma State University, Stillwater, Okla. (1958).
J. H. Whitlock, New York State Veterinary College, Cornell University, Ithaca, N.Y. (1958).
Rural Bell, School of Veterinary Medicine, Texas A. & M. College, College Station, Texas (1959).

### Poultry Diseases

C. D. Lee, *Chairman,* B-12 Curtiss Hall, Iowa State College, Ames, Iowa (1958).
J. P. Delaplane, School of Veterinary Medicine, Texas A. & M. College, College Station, Texas (1959).
Edwin Jungherr, Department of Animal Diseases, University of Connecticut, Storrs, Conn. (1959).
P. P. Levine, New York State Veterinary College, Ithaca, N. Y. (1958).
C. L. Nelson, Jewell, Iowa (1958).
F. K. Wills, Livestock Sanitary Laboratory, Salisbury, Md. (1958).

### Program*
(Ex Officio)

This committee is composed of the chairmen and secretaries of the six sections with the executive secretary or assistant executive secretary acting as the chairman (see Section Officers, adv. p. 32).

*Pursuant to article XII, section 1, part 4, of the Administrative By-Laws, as amended at the Seventy-Eighth Annual Meeting.

### Public Relations

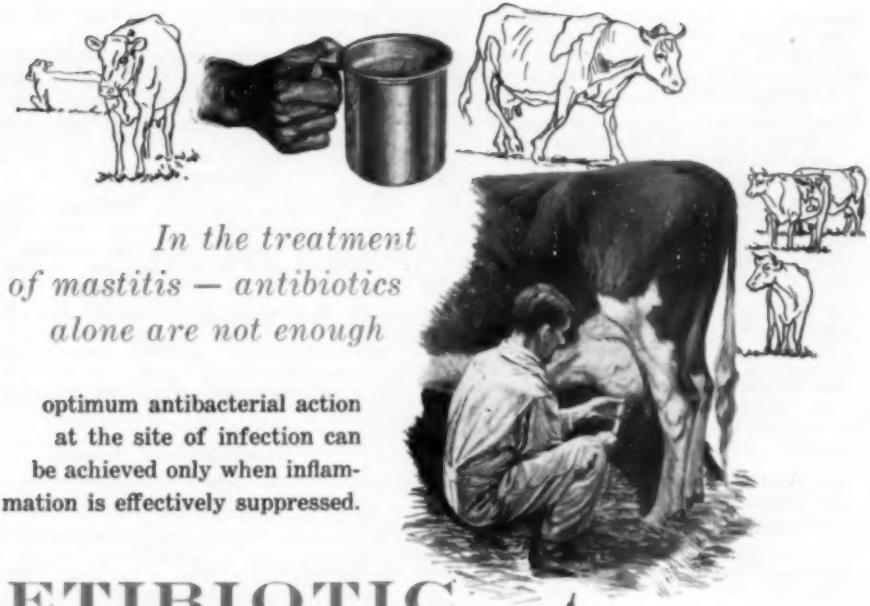
C. E. DeCamp, *Chairman,* 24 Warwick Ave., Scarsdale, N.Y. (1958).
J. R. Pickard, St. Joseph, Ill. (1958).
Dan J. Anderson, Rt. 1, Box 123, Smithfield, Texas (1958).
Kenneth G. McKay, 2086 Veterinary Science Bldg., University of California, Davis, Calif. (1958).
Erven A. Ross, 1553 44th St., N.W., Washington, D.C. (1958).
Aaron Appleby, 176 Puritan Drive, Scarsdale, N.Y. (1958).

### Registry of Veterinary Pathology
### Armed Forces Institute of Pathology

C. L. Davis, *Chairman,* 7200 E. 6th Ave., Denver Colo. (1958).
Hugh G. Grady, Scientific Director, American Registry of Pathology, Armed Forces Institute of Pathology, Washington 25, D.C. (consulting member).
T. Carl Jones, Angell Memorial Animal Hospital, 180 Longwood Ave., Boston, Mass. (1958).
C. C. Morrill, College of Veterinary Medicine, Michigan State University, East Lansing, Mich. (1959).
John H. Rust, Armed Forces Institute of Pathology, 7th St. and Independence Ave., S.W., Washington, D.C. (1958).
Marvin Twiehaus, 700 Harris St., Manhattan, Kan. (1958).

### Research Council
(Appointments are for three-year terms)

Anatomy and Histology.—Robert Getty, *Chairman,* Department of Anatomy, Iowa State College, Ames, Iowa. (1958).
Bacteriology (Immunology and Biological Therapy).—L. C. Ferguson, Head, Department of Microbiology and Public Health, College of Veterinary Medicine, Michigan State University, East Lansing, Mich. (1959).
Biochemistry and Animal Nutrition.—M. J. Swenson, Department of Physiology, College of Veterinary Medicine, Colorado State University, Fort Collins, Colo. (1959).
Large Animal Medicine.—S. J. Roberts, New York State Veterinary College, Ithaca, New York. (1959).
Large Animal Surgery.—John D. Wheat, School of Veterinary Medicine, University of California, Davis, Calif. (1960).
Parasitology.—R. D. Turk, School of Veterinary Medicine, Texas A. & M. College, College Station, Texas. (1960).



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Pathology.—T. Carl Jones, Angell Memorial Animal Hospital, 180 Longwood Ave., Boston, Mass. (1959).

Physiology and Pharmacology.—D. K. Detweiler, 48 N. Sproul Road, Broomall, Pa. (1959).

Poultry Pathology.—C. A. Brandly, College of Veterinary Medicine, University of Illinois, Urbana, Ill. (1960).

Radiology.—John H. Rust, Armed Forces Institute of Pathology, 7th St. and Independence Ave., S.W., Washington, D.C. (1960).

Small Animal Medicine.—Richard L. Ott, 1405 Gary St., Pullman, Wash. (1958).

Small Animal Surgery.—James Archibald, Ontario Veterinary College, Guelph, Ont. (1958).

Veterinary Hygiene.—A. G. Karlson, Mayo Foundation, Rochester, Minn. (1958).

Virology.—C. H. Cunningham, *Secretary*, College of Veterinary Medicine, Michigan State University, East Lansing, Mich. (1958).

Member-at-Large.—T. Lloyd Jones, Ontario Veterinary College, Guelph, Ont. (1960).

### Subcommittee on Veterinary Items, National Formulary Committee

(Ten-year appointments terminating in 1959)

B. F. Alfredson, *Chairman*, College of Veterinary Medicine, Michigan State University, East Lansing, Mich.

D. K. Detweiler, 48 N. Sproul Rd., Broomall, Pa.

Frank D. Enzie, Agricultural Research Center, U.S.D.A., Beltsville, Md.

L. Meyer Jones, Division of Veterinary Medicine, Iowa State College, Ames, Iowa.

F. J. Kingma, F.D.A., Department of Health, Education, and Welfare, Washington, D.C.

John E. Martin, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa.

### Therapeutic Agents

L. Meyer Jones, *Chairman*, Division of Veterinary Medicine, Iowa State College, Ames, Iowa. (1958).

John H. Collins, Food and Drug Administration, Department of Health, Education, and Welfare, Washington 25, D.C. (1959).

K. M. Curts, 5236 Delmar Ave., Kansas City 3, Kan. (1958).

D. K. Detweiler, 48 N. Sproul Rd., Broomall, Pa. (1958).

Roger P. Link, Department of Veterinary Physiology and Pharmacology, College of Veterinary Medicine, University of Illinois, Urbana, Ill. (1959).

W. G. Stevenson, Ontario Veterinary College, Box 220, Guelph, Ont. (1958).

## Special Committees

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A. R. Theobald, 4545 Reading Rd., Cincinnati 29, Ohio.

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Peter Olafson, New York State Veterinary College, Ithaca, N. Y.

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Karl R. Reinhard, Arctic Health Research Center, P.O. Box 960, Anchorage, Alaska.

Kenneth F. Wells, 506 Piccadilly Ave., Ottawa, Ont.

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W. J. Kilpatrick, Mediapolis, Iowa.

P. E. Madsen, 336 N. Jefferson St., Sheridan, Wyo.

J. L. McAuliff, 11 Jewett Ave., Cortland, N. Y.

A. G. Misener, 6448 N. Clark St., Chicago, Ill.

V. D. Stauffer, 5500 Wadsworth Blvd., Arvada, Colo.

Kenneth Whittington, 769 Vance Ave., Memphis, Tenn.

# COMING MEETINGS

Arkansas Veterinary Practitioners Association. Annual meeting. Velda Rose Motel, Hot Springs, Oct. 2-4, 1957. Dr. Jack R. Jones, 1115 Gee St., Jonesboro, secretary-treasurer.

West Virginia Veterinary Medical Association. Annual meeting. Daniel Boone Hotel, Charleston, West Virginia, Oct. 6-7, 1957. D. A. Munro Oglebay Hall, West Virginia University, Morgantown, secretary.

New England Veterinary Medical Association. Annual meeting. Equinox House, Manchester, Vt., Oct. 6-9, 1957. C. Lawrence Blakely, 180 Longwood Ave., Boston, Mass., secretary.

Purdue University. Annual short course for veterinarians. Purdue University, West Lafayette, Ind., Oct. 9-11, 1957. L. M. Hutchings, secretary.

Florida State Veterinary Medical Association. Annual meeting. Fort Harrison Hotel, Clearwater, Oct. 13-15, 1957. Robert P. Knowles, 2101 N.W. 25th Ave., Miami 42, Fla., secretary.

University of Missouri. Annual short course for graduate veterinarians, Oct. 14-15, 1957, School of Veterinary Medicine, University of Missouri, Columbia. Cecil Elder, chairman.

Pennsylvania State Veterinary Medical Association. Annual meeting. Hotel Brunswick, Lancaster, Oct. 16-18, 1957, Raymond C. Snyder, N. W. Corner Walnut St. and Copley Rd., Upper Darby, secretary.

Texas Veterinary Medical Association. Annual meeting. Baker Hotel, Dallas, Oct. 16-18, 1957. Paul B. Blunt, 712 Maverick Bldg., San Antonio, Texas, secretary.

Eastern Iowa Veterinary Association. Annual meeting. Hotel Sheraton-Montrose, Cedar Rapids, Oct. 17-18, 1957. F. E. Brutsman, Traer, Iowa, secretary.

Illinois, University of. Annual veterinary conference and short course. School of Veterinary Medicine, University of Illinois, Urbana, Oct. 17-18, 1957. L. E. Boley, chairman.

Southern Veterinary Medical Association. Annual meeting. Hotel Roanoke, Roanoke, Va., Oct. 27-30, 1957. A. A. Husman, P. O. Box 91, Raleigh, N. Car., secretary.

Cornell University. Nutrition conference. Cornell University, Ithaca, N.Y., Oct. 31-Nov. 1, 1957. J. K. Loosli, Stocking Hall, Cornell University, Ithaca, N.Y., chairman.

Interstate Veterinary Medical Association. Annual meeting. Martin Hotel, Sioux City, Iowa, Nov. 7-8, 1957. K. W. Smith, 1002 34th St., Sioux City, Iowa, secretary.

Animal Care Panel. Annual meeting. Bellevue Hotel, San Francisco, Calif., Nov. 7-9, 1957. R. J. Flynn, Box 299, Lemont, Ill.

Pennsylvania, University of. Annual conference for veterinarians. School of Veterinary Medicine, Philadelphia, Jan. 7-8, 1958. M. W. Allam, dean.

Cornell University. Annual conference for veterinarians. New York State Veterinary College, Ithaca, Jan. 8-10, 1958. W. A. Hagan, dean.

Oklahoma Veterinary Medical Association. Annual meeting. Hotel Lawtonian, Lawton, Jan. 9-10, 1958. Mrs. Larma Bennett, 2805 S. W. 51 St., Oklahoma City, executive secretary.

Kansas Veterinary Medical Association. Annual convention. Hotel Broadview, Wichita, Jan. 12-14, 1958. K. Maynard Curts, 5236 Delmar Ave., Kansas City 3, Kan., secretary.

Iowa Veterinary Medical Association. Annual meeting.


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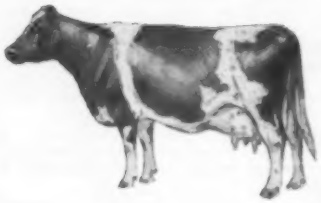

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Hotel Fort Des Moines, Des Moines, Jan. 14-16, 1958. F. B. Young, Waukee, Iowa, secretary.

Intermountain Veterinary Medical Association. Annual meeting. Hotel Utah, Salt Lake City, Jan. 16-18, 1958. R. A. Bagley, 4600 Creek View Dr., Murray, Utah, secretary.

Minnesota Veterinary Medical Association. Annual meeting, St. Paul Hotel, St. Paul, Jan. 20-22, 1958. B. S. Pomeroy, School of Veterinary Medicine, University of Minnesota, St. Paul 1, Minn., secretary.

North Carolina State College. Conference for veterinarians. North Carolina State College, Raleigh, Jan. 28-31, 1958. C. D. Grinnells, chairman.

Oregon Veterinary Medical Association. Winter meeting. Portland, Jan. 31-Feb. 1, 1958. Edward L. Holden, P. O. Box 445, Oswego, secretary.

## Foreign Meetings

Seventh Brazilian Congress of Veterinary Medicine. Recife, Pernambuco, Brazil, Oct. 12-19, 1957. Dr. Valdi Moreire Martius, Caixa Postal 2505, Recife, secretary-general.

## Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Association, the first Thursday of each month. B. M. Lauderdale, Montgomery, secretary.

Jefferson County Veterinary Medical Association, the second Thursday of each month. S. A. Price, 213 N. 15th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the third Tuesday of each month. W. David Gross, 771 Holcombe Ave., Mobile, Ala., secretary.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2, Box 697, Tucson, Ariz., secretary.

CALIFORNIA—Alameda-Contra Costa Veterinary Medical Association, the fourth Wednesday of Jan., March, May, June, Aug., Oct., and Nov. Leo Goldston, 3793 Broadway, Oakland 11, Calif., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of February, April, July, September, and December at 3004 16th St., San Francisco, Calif. Mr. Herb Warren, executive secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. R. B. Barsuleau, 2333 E. Mineral King, Visalia, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of each month. A. L. Irwin, 301 Taft Highway, Bakersfield, Calif., secretary.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Caveil, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2643 N. Main St., Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of each month. R. C. Lawson, 4040 El Camino, Palo Alto, Calif., secretary.

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Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert E. Clark, Napa, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. John Chudacoff, 7912 Sepulveda Blvd., Van Nuys, secretary.

Santa Clara Valley Veterinary Association, the fourth Tuesday of each month. Kay Beulley, N. Fourth and Gish Rd., San Jose, Calif., secretary.

Southern California Veterinary Medical Association, the last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinarians, the second Thursday of each month. R. B. Barsalesu, 2333 E. Mineral King, Visalia, Calif., secretary.

COLORADO—Denver Area Veterinary Society, the fourth Tuesday of every month. Richard C. Tolley, 5060 S. Broadway St., Englewood, Colo., secretary.

Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammarlund, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

FLORIDA—Central Florida Veterinary Medical Association, the first Tuesday of each month, time and place specified monthly. Jack H. McElyea, 5925 Edgewater Drive, Orlando, Fla., secretary.

Florida West Coast Veterinary Medical Association, the second Wednesday of each month at the Lighthouse Inn, in St. Petersburg. R. L. Brutus, 336 E. 15th St., Hialeah, Fla., secretary.

Jacksonville Veterinary Medical Association, the first Thursday of every month. Dodsons Restaurant. P. S. Roy, 4443 Atlantic Blvd., Jacksonville, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. T. R. Geci, 108B Catherine Ave., Pensacola, Fla., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 810 Datura St., West Palm Beach. J. J. McCarthy, 500-25th Street, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Wednesday of each month. Time and place specified monthly. Frank Mueller, Jr., 4148 E. 8th Ave., Hialeah, Fla., secretary.

Suwannee Valley Veterinary Association, the fourth Tues-

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Volusia County Veterinary Medical Association, the fourth Thursday of each month. A. E. Hixon, 131 Mary St., Daytona Beach, Fla., secretary.

GEORGIA—Atlanta Veterinary Society, the second Tuesday of every month at the Elks Home on Peachtree St., Atlanta, Ga. J. L. Christopher, Smyrna, Ga., secretary.

ILLINOIS—Chicago Veterinary Medical Association, the second Tuesday of each month. Mark E. Davenport, Jr., 215 S. Edgewood Ave., LaGrange, Ill., secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. H. S. Bryan, College of Veterinary Medicine, University of Illinois, Urbana, secretary.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary.

Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at the Hotel LaSalle, South Bend, Ind. J. M. Carter, 3421 S. Main St., Elkhart, Ind., secretary.

Tenth District Veterinary Medical Association, the third Thursday of each month. J. S. Baker, P. O. Box 52, Pendleton, Ind., secretary.

IOWA—Cedar Valley Veterinary Association, the second Monday of each month, except January, July, August, and October, at Black's Tea Room, Waterloo, Iowa. H. V. Henderson, Reinbeck, Iowa, secretary.

Coon Valley Veterinary Association, the second Wednesday of each month, September through May, at the Bradford Hotel, Storm Lake, Iowa. D. I. Lee, Sac City, Iowa, secretary.

East Central Iowa Veterinary Medical Society, the second Tuesday of every month. Dr. W. T. Rugger, Oxford, secretary.

Fayette County Veterinary Association, the third Tuesday of each month, except in July and August, at Pa and Ma's Restaurant, West Union, Iowa. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Wisneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

KANSAS—Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Tuesday of each month. Robert E. Guilfoil, 18 N. 2nd St., Kansas City 18, Kansas, secretary.

KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. L. S. Shirrell, Versailles Rd., Frankfort, secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday evening of each month in Louisville or within a radius of 50 miles. W. E. Bewley, P.O. Box "H," Crestwood, secretary.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m. at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Harry L. Schultz, Jr., 9011 Harford Rd., Baltimore, Md., secretary.

MICHIGAN—Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May.


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REFERENCES: 1. Master, J. E.: Vet. M. 50:605 (Nov.) 1955. 2. Belloff, G. B.: Calif. Vet. 9:27 (Sept.-Oct.) 1956. 3. Pollock, S., J. Am. Vet. M. Ass. 129:274 (Sept.) 1956.

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# What Is Your Diagnosis?

A case history and radiograph depicting a diagnostic problem appear below.

**Make your diagnosis from the picture below—then turn the page ▶**

Figure 1

*History:*—A male cat, 2 years old, developed generalized soreness over the entire body but showed no signs of injury. The soreness seemed mild but the cat hesitated to move and it would cry and bite when picked up. There was a persistent mild fever (102.3 F.) but the appetite remained good.

The general soreness soon disappeared but the cat continued to limp on the right foreleg and kept the leg flexed when it jumped. A radiograph was taken of the right forelimb.

## Here Is the Diagnosis

*(Continued from preceding page)*

*Diagnosis.*—Multicentric osteomyelitis of the right humerus of a cat.

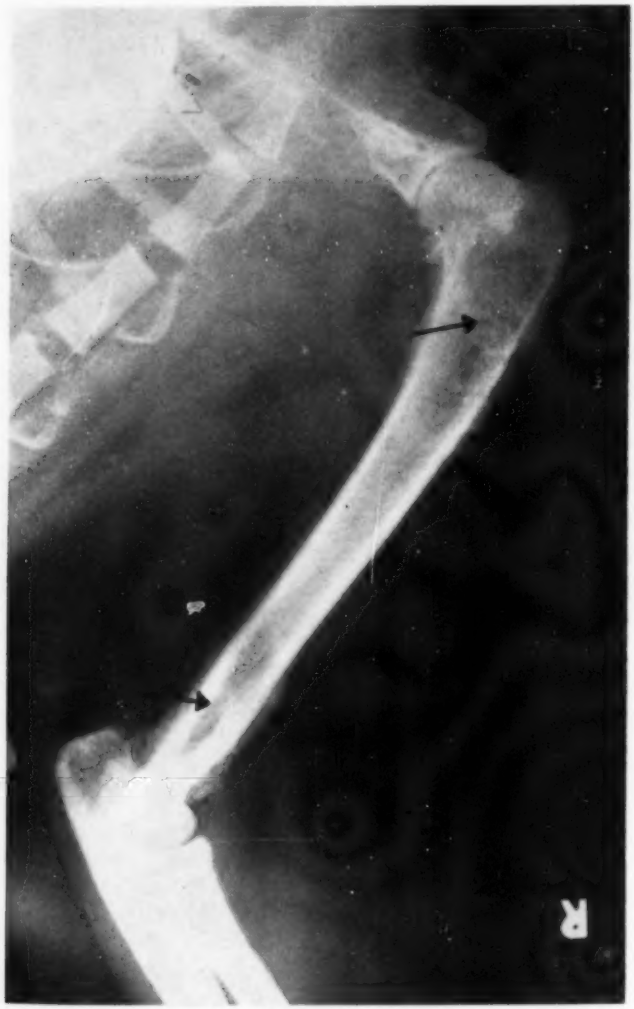

**Fig. 2—Radiograph of the right humerus showing the lesions (arrows) of multicentric osteomyelitis.**

*Comment.*—Radiographically, this is a picture of bone proliferation and destruction. The middle portion of the shaft (fig. 2) is not affected. Osteomyelitis of this type is always hematogenous, the pyogenic organisms (usually *Staphylococcus aureus,* in man) becoming lodged in capillaries at the metaphysis at one or both ends of the bone. Here, especially in the young animal, the bone is soft and cancellous. As the organisms multiply, the resulting lesions spread along the area of least resistance up and down the medullary canal and may perforate the compact bone at widely separated points, depending upon its density. When the periosteum becomes involved, it forms a new layer of bone known as an involucrum.

This infection responded promptly, as have others of this type, to antibiotic therapy. When infection is eliminated, the lesions usually disappear and the bone then appears normal.

---

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Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of each month (except July and August), at the Coronado Hotel, Lindell Blvd. and Spring Ave., St. Louis, Mo., at 8 p.m. Chester R. Plegge, 4249 Peck St., St. Louis 7, Mo., secretary.

Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Tuesday of each month. Robert E. Guilfoil, 18 N. 2nd St., Kansas City 18, Kansas, secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Hightstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Milburn Ave., Maplewood, N. J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Casa Mana in Teaneck. James R. Tanzola, Upper Saddle River, secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. G. R. Muller, 43 Church St., Lambertville, N. J., secretary.

Southern New Jersey Veterinary Medical Association, the third Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. W. E. Snyder, E. Kings Highway and Munn Ave., Haddonfield, secretary.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

New York State Veterinary College. Annual conference for veterinarians. Cornell University, Ithaca. W. A. Hagan, New York State Veterinary College, Cornell University, Ithaca, N. Y., dean.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. Joseph A. Lombardo, 411 Woodlawn Ave., Greensboro, secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month, time and place specified monthly. Byron H. Brow, Box 453, Goldsboro, N. Car., secretary.

Piedmont Veterinary Medical Association, the last Friday of each month. John G. Martin, Boone, N. Car., secretary.

Twin Carolinas Veterinary Medical Association, the third Thursday of each month in the Orange Bowl Restaurant, Rockingham, N. Car., at 7:30 p.m. James R. Burgess, Rockingham, N. Car., secretary.

OHIO—Cuyahoga County Veterinary Medical Association, the first Wednesday of each month, September through May (except January), at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. Ed. R. Jacobs, 5522 Pearl Rd., Cleveland, Ohio, secretary.

Stark County Veterinary Association, the second Monday of each month. M. L. Willen, 4423 Tuscarawas St., Canton, Ohio, secretary.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month, 7:30 p.m., Patrick's Foods Cafe, 1016 N.W. 23rd St., Oklahoma City. Forrest H. Stockton, 2716 S.W. 29th St., Oklahoma City, Okla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Don L. Hohmann, 538 S. Madison St., Tulsa, Okla., secretary.

OREGON—Willamette Veterinarian Medical Association, the third Tuesday of each month, except July and August, at the Marion Hotel, Salem. Dr. Marvin Corff, McMinnville, secretary.

PENNSYLVANIA—Del-High Veterinary Medical Association, the first Thursday of each month. Stewart Rockwell, 10th and Chestnut Sts., Emmaus, Pa., secretary.

Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine, 39th and Woodland Ave., Philadelphia 4, Pa. Raymond C. Snyder, 39th and Woodland Ave., Philadelphia 4, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

VIRGINIA—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary St., Richmond 20, Va., secretary.

Northern Virginia Veterinary Conference, the second Tuesday of each month. Francis E. Mullen, 1130 S. Main St., Harrisonburg, Va., secretary-treasurer.

Northern Virginia Veterinary Society, the second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, secretary

Southwest Virginia Veterinary Medical Association, the first Thursday of each month. I. D. Wilson, Blacksburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Monday of each month, Magnolia American Legion Hall, 2870 32nd W., Seattle, Wash. William S. Green, 9637 S. E. 36th, Mercer Island, Wash., secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. O. L. Bailey, P. O. Box 906, Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Prichard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

Central Wisconsin Veterinary Medical Association, the second Tuesday of each quarter (March, June, Sept., Dec.). R. J. O'Hern, P. O. Box 617, Cumberland, Wis., secretary.

Dane County Veterinary Medical Association, the second Thursday of each month. Dr. E. P. Pope, 409 Farley Ave., Madison, Wis., secretary.

WISCONSIN—Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. Dr. R. H. Steinkraus, 7701 N. 59th St., Milwaukee, Wis., secretary.

Northeastern Wisconsin Veterinary Medical Association, the third Wednesday in April. William Madison, 218 E. Washington St., Appleton, Wis., secretary.

Rock Valley Veterinary Medical Association, the first Wednesday of each month. W. E. Lyle, P. O. Box 107, Deerfield, Wis., secretary.

Southeastern Veterinary Medical Association, the third Thursday of each month. John R. Curtis, 419 Cook St., Portage, Wis., secretary.

Wisconsin Valley Veterinary Medical Association, the second Tuesday of every other month. E. S. Scobell, Rt. 2, Wausau, Wis., secretary.

## AVMA Research Fellowships Available

The Research Council of the AVMA announces the availability of a number of fellowships for postgraduate training for the academic year, 1958-1959.

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The latest date for filing the completed application is Feb. 15, 1958. Approximately one month is required for processing completed applications after receipt by the secretary of the Council. Qualified persons should secure and submit applications as early as possible to insure their file being complete for presentation to the Committee on Fellowships.

The Committee on Fellowships of the Research Council will meet in March to consider applications, and the awards will be announced soon afterward. The stipend will be determined in each case by the needs of the individual, the location of the school in which he proposes to work, and other factors. In general, the stipends range from $100 monthly and upward.

Any qualified person interested in graduate training may obtain application blanks and other information by writing to Dr. C. H. Cunningham, secretary, AVMA Research Council, Department of Microbiology and Public Health, College of Veterinary Medicine, Michigan State University, East Lansing, Mich.

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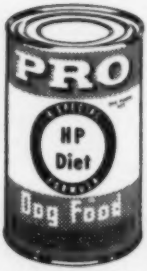

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Veterinarian wanted for general practice in Maryland. 75% dairy practice with large and small animal hospital facilities. Salary or commission. Could lead to partnership. State experience, references, salary expected. Dr. F. D. Custer, Oakland, Md.

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(*Classified ads continued on p. 57*)

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Report of the Committee on Diseases of Dairy Cattle, R. C. Klussendorf, Chairman. J.A.V.M.A. *107*:355 (Nov.) 1945.

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[illegible], R. T., Newton, D. L., and Huber, W. G.: [illegible] presented at 93rd Annual Meeting, A.V.M.A., Dec. 10-13, 1956, San [illegible].

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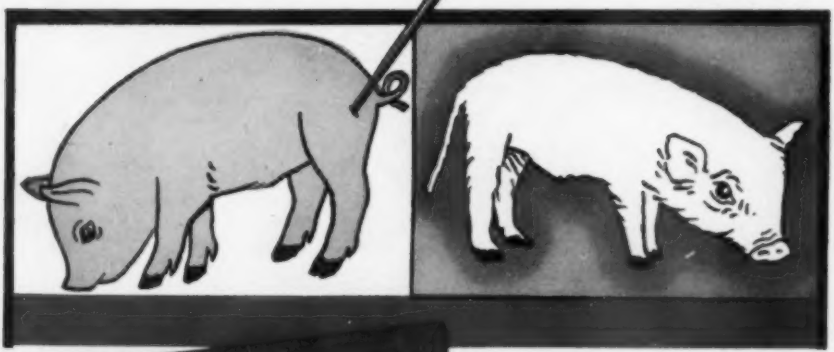

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